Department of Health

Report on Health and Social Subjects

34

Report on Confidential Enquiries into Maternal Deaths in England and Wales 1982–1984

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Preface

CEMD Report 1982–84

This is the eleventh report in the series on maternal deaths and is number 34 of the "Reports on Health and Social Security" published under the auspices of the Department of Health. After 33 years, it is the last of the series for England and Wales alone, because for the next period from 1985 it is intended that a combined United Kingdom report will be published to include also maternal deaths in Scotland and Northern Ireland. It can therefore be regarded as a milestone in a historic series and for this reason the authors decided that it should include an "Overview" of the reports since 1952–54.

I am most grateful to Sir Alexander Turnbull for writing this additional Chapter (19) for the report. He has illustrated the interesting fact [Fig 19.1] that the maternal mortality rate has approximately halved every 10 years over the last 50 years, and if these rates are expressed on a logarithmic scale, the reduction in maternal mortality rate is almost linear, indicating that the *rate* of fall has been constant. The number of deaths has, of course, fallen very substantially over this period and continues to fall. He has also included a brief survey of maternal mortality since 1847, putting the series of reports into historical perspective.

In this report 243 deaths remained after the 73 late deaths, (ie those occurring more than 42 days after pregnancy and delivery), had been excluded. The late deaths have been described in a separate chapter (Chapter 16). Of the 243 deaths, 138 were Direct obstetric deaths, 71 Indirect obstetric deaths and 34 were Fortuitous (ie considered to be unrelated to the pregnancy). Indirect obstetric deaths have become relatively of more importance and greater interest with the decline in the number of Direct obstetric deaths. As an indication of the increased importance of many of these Indirect deaths, from such causes as epilepsy and aneurysm, the authors have described some of them in detail in Chapter 14 and separated them from the Fortuitous deaths, listed in Chapter 15.

The report includes over 98 per cent of the known Direct obstetric deaths. One death from eclampsia was not reported, and a death from legal abortion was reported too late to be included in the figures but has been described as an addendum to the chapter on Abortion (Chapter 11).

A notable feature of the report compared with earlier reports, from the years before the Abortion Act (1967) came into force, is that for the first time in this triennium, there was no maternal death due to criminal abortion, compared with 75 to 80 deaths in similar periods only 20 years previously.

Pulmonary embolism and hypertensive diseases of pregnancy were the commonest causes of maternal death, during the triennium, with anaesthesia and amniotic fluid embolism third and fourth. Overall in 52 per cent of the Direct obstetric deaths some aspects of the care of the patient was found to be "substandard" by the assessors. This term is used to include not only failures of clinical care but also shortage of staff or facilities or administrative failure in the maternity or back-up services.

Chapter 18 of the report includes the recommendations made by the assessors for improvements in the maternity services and in the treatment of individual women. They found that the highest proportion of cases with substandard care were in women with hypertensive diseases of pregnancy and in the anaesthetic deaths. Particular emphasis was laid on the importance of the effective control of the severe hypertension which is the cause of the high proportion of deaths from intracranial haemorrhage in women with eclampsia and pre-eclampsia. The number of anaesthetic deaths has fallen despite the increased number of anaesthetics given to pregnant women. Probably the most important recommendation, for further reducing the number of anaesthetic deaths, is the one for better consultation and planning between the obstetric and anaesthetic teams for the benefit of women suspected to be at high risk. This should minimise the number of occasions when a junior anaesthetist has to deal unexpectedly with difficult problems which should have been foreseen.

Deaths from pulmonary and amniotic fluid embolism seem at the moment to be the most difficult in which to effect an improvement. However an increased awareness of the possibility of thrombosis and pulmonary embolism in pregnancy, labour and the puerperium could be expected to produce earlier diagnosis and more effective treatment. Amniotic fluid embolism awaits a breakthrough in its prevention, detection and treatment; only the first positive step, the accurate diagnosis of the condition at autopsy, has so far been taken.

Despite the increase in the number of Caesarean sections performed, a trend which was also occurring in other Western countries during the triennium, the actual number of deaths associated with Caesarean section fell. The fatality rate for all deaths following Caesarean section in NHS hospitals in England and Wales continued to decline being 0·37 per 1000 Caesarean sections compared with 0·52 (1979–81) and 0·66 (1976–78).

Similarly, there was a fall in the number of deaths from ectopic pregnancy. The death rates fell from 8·2 per million estimated pregnancies in 1979–81 to 4·1 in 1982–84. Again this fall in mortality occurred despite an increase in the incidence of ectopic pregnancy in the population.

The numbers of deaths from haemorrhage and from genital sepsis fell once more in this triennium. However deaths from haemorrhage remained an important factor in maternal mortality, and the authors have again included suggested guidelines for obstetric units, for an agreed procedure for the management of massive haemorrhage. These are listed at the end of Chapter 3.

Most of the Tables in this report have been made to include the fifteen years from the 1970–72 triennium. This was the earliest period from which it was possible to exclude the late deaths and in which the proportion of deaths and death rates from the main causes was reasonably comparable with more recent triennia [Tables 19.3 and 19.5].

I would like to thank the obstetricians, anaesthetists, pathologists, district medical officers, coroners, general practitioners and midwives who over the years have contributed so much to the individual case reports. I am also most grateful for the work of the Regional Assessors in Obstetrics, Anaesthetics and Pathology who have given a great deal of their time and expertise to the assessment of the cases. The Regional Obstetric Assessors have met with the authors of the report under the chairmanship of Dr Michael Abrams, a Deputy Chief Medical Officer, to discuss the contents of the report and the Regional Anaesthetic and Pathology Assessors have commented on the anaesthetic and pathology chapters. The final assessments and reports take into account the views expressed by the Regional Assessors and these are coordinated by the authors to produce a report which is generally acceptable to all who contributed.

My principal acknowledgments and thanks go to the main authors of the report, my former Consultant Adviser in Obstetrics and Gynaecology, Sir Alexander Turnbull and my present Consultant Advisers, Professor V R Tindall and Professor R W Beard, and the former Consultant Advisers in Anaesthetics, Sir Gordon Robson and in Histopathology, Professor I M P Dawson. They have had the help of Dr John Ashley, Mrs Beverley Botting and the other staff of the Medical Statistics Unit of the Office of Population, Censuses and Surveys, Miss Alison Macfarlane of the National Perinatal Epidemiology Unit, Dr Elizabeth Cloake of the Department of Health and Dr Mary Cotter of the Welsh Office.

Three of the authors are now retiring after a long and fruitful association with the Confidential Enquiries, and I would like to express my particular appreciation for their work. Sir Alexander Turnbull has been associated with the enquiry since 1973 first as a Regional Assessor and then as a Central Obstetric Assessor for four triennia. In addition to his work on the present report, he has also contributed from the basis of his long experience the 'Overview' of the series and has provided a novel way of looking at the significance of maternal mortality rates. Sir Gordon Robson was the Central Assessor for anaesthetics for the three triennia 1973–75, 1976–78 and 1979–81. In 1986 he very kindly agreed to return to complete the work on a fourth, the 1982–84 report, following the sad, early death of Professor Cyril Conway. The maternal deaths associated with anaesthesia have decreased in number over the triennia but have become the third main cause as other Direct causes of death have declined more rapidly. By careful assessment of the problems Sir Gordon has illuminated the need for improved standards of anaesthetic care for all maternity patients. Professor Ian Dawson was appointed in 1980 as the first Central Assessor in Pathology to the enquiries. He was concerned with the 1976–78 report and then with the appointment of the first Regional Assessors in Pathology in 1981. With their help he has influenced and encouraged the considerable improvement in the standards of autopsies and their reporting which has been evident in the 1979–81 and 1982–84 reports.

Finally, I should now look ahead to the future, and wish success to the Assessors and medical officers in the four countries who are working together to produce a combined United Kingdom report for the 1985–87 maternal deaths. I hope that this will be the first of a continuing successful series of medical audits. If Sir Alexander Turnbull's predictive graph continues its long term trend we can hope for a further decline in substandard care and maternal deaths over the next decades.

E D ACHESON
Chief Medical Officer

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1. Introduction

Trends in maternal mortality

This is the eleventh triennial report of Confidential Enquiries into maternal deaths. It extends the coverage of these reports to a period of more than 30 years during which more than 24 million births have been registered in England and Wales. Additionally, between the introduction of the 1967 Abortion Act in 1968 and the end of 1984 nearly 1.8 million legal terminations of pregnancy have been carried out on residents of England and Wales.

This chapter presents explanatory statistics drawn from official sources to place in context the maternal deaths; Tables 1.1 to 1.4 use data drawn exclusively from official sources and not from the enquiry.

From Table 1.1 it can be seen that the number of births and the birth rate in the 1982–1984 triennium were lower than those experienced in the previous triennium. During the same three years the number of legal abortions was almost 4 per cent higher than in the previous triennium (Table 1.2). However, since the number of women aged 15 to 44 also rose between 1979–81 and 1982–84, the abortion rate in 1982–1984 per 1,000 women aged 15 to 44 was less than one per cent higher than in 1979–1981.

Table 1.3 shows statistics for every third triennium between 1955–1957 and 1982–84. Over this period the maternal mortality rate fell from 69·1 to 8·6 per 100,000 total births, roughly halving between each given triennium; in comparison, the general fertility rate in 1982–84 (60·1 births per 1,000 women aged 15–44) was only slightly lower than the rate in 1955–57 (78·4).

Table 1.1 *Total number of births (live and still), 1982–1984, England and Wales*

Year of occurrence	Total births	Rate per 1000 women aged 15–44
1952–1954	2,079,275	74·4
1955–1957	2,140,376	78·4
1958–1960	2,322,229	85·7
1961–1963	2,550,252	92·1
1964–1966	2,630,150	92·8
1967–1969	2,484,004	88·0
1970–1972	2,322,124	82·3
1973–1975	1,940,689	67·9
1976–1978	1,766,169	60·1
1979–1981	1,942,859	63·4
1982–1984	1,903,096	60·1

Source: Birth Statistics, Series FM1 No 11 Table 3.2
FM1 No 13 Table 1.1, 1.2

Table 1.2 *Legal abortions to women resident in England and Wales (1970–84)*

Triennia	No. of abortions	Rate per 1000 women aged 15–44
1970–1972	279,097	9·9
1973–1975	326,237	11·4
1976–1978	316,440	10·8
1979–1981	378,119	12·3
1982–1984	392,316	12·4

Source: Abortion Statistics, Series AB No 11 Table 2.

Table 1.3 *Some statistics of maternal mortality and births 1955–1984, England and Wales*

	1955–57†	1964–66‡	1973–75*	1982–84**
Number of maternal deaths:				
Total	1480	830	323	163
of which				
Abortion	199	155	31	20
Direct obstetric deaths	913	516	215	128
Associated obstetric deaths	368	159	77	15
Total births	2,140,376	2,630,150	1,940,689	1,903,096
Maternal mortality rate per 100,000 total births	69·1	31·6	16·6	8·6
General fertility rate per 1000 Women aged 15–44 years	78·4	92·8	67·9	60·1

† ICD 6th Revision
‡ ICD 7th Revision
* ICD 8th Revision
** ICD 9th Revision

Source: 1955–75 Mortality statistics, childhood and maternity, Series DH3, No 4, Table 18.
1982–84 Mortality statistics, cause, Series DH2, Table 2.
Mortality statistics, Series DH1, Table 25
1955–83 Birth statistics, Series FM1, No 11, Table 1.1, 1.2.
1984 Birth statistics, Series FM1, No 13, Table 1.1, 1.2

Table 1.4 *Mortality rates per million population for females aged 15–44 years: All causes and maternal deaths (1952–1984), England and Wales*

Triennia	Mortality rates per million females aged 15–44 years		
	All causes	Maternal deaths	% of deaths in the age-group that are maternal
1952–1954	1275·3	50·3	3·9
1955–1957	1125·0	40·7	3·6
1958–1960	1016·8	34·3	3·4
1961–1963	988·1	29·5	3·0
1964–1966	950·0	23·7	2·5
1967–1969	879·6	18·7	2·1
1970–1972	831·9	13·9	1·7
1973–1975	787·0	8·6	1·1
1976–1978	741·2	7·5	1·0
1979–1981	682·4	6·6	1·0
1982–1984	627·8	4·7	0·7

Source: The Registrar General's Statistical Review of England and Wales, Part 1; Mortality Statistics, cause, Series DH2, Table 2.

For women aged 15–44 maternal mortality rates have fallen faster than the death rates from all causes for women in the same age group. Table 1.4 shows that in 1982–84 maternal deaths formed less than one per cent of all deaths of women in this age group, compared with almost 4 per cent in 1952–54.

The 1982–84 enquiry

The total number of maternal deaths reported to the enquiry for 1982–1984 is consistent with a continued reduction in maternal mortality. There were 316 deaths known to the enquiry, of which 243 (77 per cent) occurred during pregnancy or before 42 days postpartum (the international definition of maternal death). The remaining 73 deaths for this triennium are classed as 'late' deaths and are considered separately in Chapter 16. The total number of maternal deaths in 1982–1984 represents a fall of 13 per cent compared with 1979–81, when excluding the late deaths, the number in pregnancy and puerperium had fallen by 19 per cent.

In accordance with the practice established in the last two reports, maternal deaths are subclassified into 'Direct' deaths resulting from obstetric complications of pregnancy, labour and the puerperium, 'Indirect' deaths resulting from previous existing disease or a disease which developed during pregnancy and which was aggravated by pregnancy, and 'Fortuitous' deaths resulting from causes not related to or influenced by pregnancy.

Table 1.5 shows that the 243 maternal deaths in 1982–84 were classified as 138 (57 per cent) Direct, 71 (29 per cent) Indirect, and 34 (14 per cent) Fortuitous. This compares with 176 Direct (59 per cent), 92 Indirect (31 per cent) and 31 Fortuitous (10 per cent) maternal deaths in the previous triennium.

Table 1.5 *Reported deaths in relation to stage of pregnancy at which death occurred, 1982–84*

	Direct deaths	Indirect deaths	Fortuitous deaths	Total
Total (excluding late deaths)	138	71	34	243
Deaths under 28 weeks gestation	39	17	15	71
of which: After abortion	15†	4	4	23
Ectopic pregnancy	13*	1	—	14
Undelivered	9	11	11	31
Delivery of liveborn	2	1	—	3
Deaths from 28 weeks gestation to 6 weeks postpartum	99	54	19	172
of which: Undelivered	17	12	7	36
Delivered	82	42	12	136
Deaths later than 6 weeks postpartum	6	17	50	73

† Includes 4 deaths not included in the abortion chapter: 2 deaths from anaesthesia and 2 deaths from hypertensive disease following abortion.

* Includes 3 deaths from anaesthesia not included in the chapter on ectopic pregnancies.

Table 1.5 also shows that 71 women died before the 28th week of their pregnancy; this proportion of the 243 maternal deaths (29 per cent) being slightly higher than in the previous two triennia. There were 3 maternal deaths before 28 weeks gestation associated with live births.

The wide regional variation in maternal mortality is shown in Table 1.6 in which the period 1976–1984 is used to increase the number of maternal deaths available for analysis. Over this period the direct obstetric maternal mortality rate (maternal deaths per 100,000 total births) varied between 6·1 per 100,000 births in South Western RHA and 12·4 per 100,000 births in North East Thames RHA. However, this variation was smaller than that shown in a similar analysis based on 1976–1981 given in the previous report (where the rates varied from 5·5 to 16·1 per 100,000 births). There was also considerable regional variation in the ratio of Direct to Indirect deaths.

Table 1.6 also shows the perinatal mortality rates for each RHA over the same period. However, Kendall's rank correlation test showed no significant correlation between the ranked order of the direct obstetric mortality rate and that of the perinatal mortality rate.

Table 1.6 *Reported direct and indirect maternal mortality by Regional Health Authority of residence, 1976–1984*

Regional Health Authority	Reported			Direct obstetric mortality rate**	Perinatal mortality rate†
	Total births	Direct deaths*	Indirect deaths		
Northern	354,326	43	17	12·1	14·8
Yorkshire	410,541	36	26	8·8	14·8
Trent	511,908	52	23	10·2	13·5
East Anglian	208,780	25	9	12·0	11·5
North West Thames	408,329	36	12	8·8	11·9
North East Thames	442,293	55	7	12·4	12·9
South East Thames	392,622	31	9	7·9	13·1
South West Thames	304,857	19	11	9·5	11·7
Wessex	293,272	20	11	6·8	11·7
Oxford	278,521	20	10	7·2	11·4
South Western	328,257	20	17	6·1	12·5
West Midlands	604,229	66	24	10·9	15·6
Mersey	281,491	28	19	9·9	13·7
North Western	470,946	49	24	10·4	14·7
Wales	317,323	23	20	7·2	14·3
England and Wales***	5,612,124	533	239	9·5	13·5

* Includes abortion
** Per 100,000 total births
*** Includes women normally resident outside England & Wales
† Stillbirths & deaths under 1 week combined per 1000 live and stillbirths

Further characteristics of births

In accordance with established custom, in most of the succeeding chapters of this report, maternal deaths will be related to the number of maternities (mothers delivered of live or stillborn infants) rather than to the number of births, since this latter statistic is slightly inflated by multiple births. The combination of the numbers of maternities together with legal terminations, with an adjustment to allow for the period of gestation and maternal ages at conception, provide data which are now referred to in official statistics as 'conceptions'. These statistics are based on the date of conception. This explains the differences between these data and those given in previous tables in this chapter which are based on the date of delivery.

Between 1974 and 1984 the number of conceptions outside marriage to women in their twenties and thirties almost doubled. However, this was largely due to the increasing population of unmarried women of these ages since the conception rates per 1000 unmarried women changed relatively little over the period. The proportion of conceptions which were terminated by legal abortion, both those inside and those outside marriage, remained stable between 1974 and 1984, at fewer than 10 per cent of conceptions inside marriage and just under 40 per cent of all conceptions outside marriage.

In Table 1.7 an estimate has been made of the number of 'pregnancies'. The number of hospital admissions for spontaneous abortions (up to 28 weeks gestation) and ectopic pregnancies are added to the official estimates of the number of 'conceptions'. This total is clearly an underestimate of the actual number of pregnancies since not all women who abort are admitted to hospital.

The estimated number of pregnancies in 1982–1984 was 1·4 per cent lower than in 1979–81. This is largely accounted for by a fall of 15·4 per cent in the number of hospital admissions for spontaneous abortions. However, there were increases in the number of legal abortions and ectopic pregnancies by 3·3 per cent and 19·0 per cent respectively.

Table 1.8 shows the estimated number of pregnancies by the age of the mother together with the percentage change from 1979–81 to 1982–84.

Table 1.7 *Estimated number of pregnancies 1970–1984 residents of England and Wales (thousands)*

	Estimated conceptions		Hospital admissions		Total estimated pregnancies
	Maternities	Legal abortions	Spont. abortions	Ectopic pregnancies	
1970–1972	2,222·5	290·9	†207·6	11·6	2,890·7
1973–1975	1,851·9	327·9	†175·3	11·7	2,578·4
1976–1978	1,781·3	324·6	†158·3	11·6	2,323·8
1979–1981	1,910·9	380·5	*134·3	12·1	2,543·2
1982–1984	1,905·8	393·1	*113·6	14·4	2,507·0

† ICD (8th revision) 640–645
* ICD (9th revision) 634–638

Source: Birth statistics 1837–1983, Series FM1, No 13, Table 12.2
Birth statistics 1984, series FM1, No 11.
Hospital In-patient Enquiry

Note: Conception statistics are based on date of conception

Table 1.8 *Estimated number of pregnancies to residents of England and Wales (thousands), 1970–1984*

Triennia	Number of pregnancies			
	All ages	24 years and under	25–34 years	35 years and over
1970–1972	2,890·7	1,478·2	1,186·9	225·6
1973–1975	2,578·4	1,242·9	1,153·6	182·0
1976–1978	2,323·0	1,070·1	1,099·9	152·9
1979–1981	2,543·2	1,176·0	1,189·1	178·1
1982–1984	2,507·0	1,147·2	1,164·9	194·9
Percentage change 1979–1981 to 1982–1984	−1·4	−2·4	−2·0	+9·4

The demographic characteristics of maternal deaths

Country of birth

Within the general decline in the number of births and maternal deaths, there was variation according to country of birth. Compared to the previous triennium, the number of total births in 1982–1984 to mothers born outside the United Kingdom fell more than those to mothers born within the United Kingdom. As a result, their share of live births fell to 12·7 per cent in 1982–1984. However, it is important to note that birthplace does not necessarily equate with ethnic origin. In particular, there is an increasing

Table 1.9 *Total births and maternal deaths by birth-place of mother, 1982–84 compared with 1979–81 (in parentheses)*

Birth-place of mother	Number of total births (1000s)	Number of maternal deaths	Ratio of maternal deaths to total births (per million)	Perinatal mortality rate*
Total	1,903·1 (1,942·9)	209 (268)	109·8 (137·9)	10·5 (13·2)
United Kingdom	1,659·8 (1,686·6)	173 (211)	104·2 (125·1)	10·2 (12·9)
Total Outside UK	242·7 (255·2)	36 (53)	148·3 (207·7)	12·3 (15·2)
Irish Republic	20·6 (27·3)	1 (1)	48·5 (36·6)	9·6 (14·4)
Australia, Canada, New Zealand	6·8 (7·3)	– (2)	– (274·0)	6·9 (9·3)
New Commonwealth	118·3 (122·2)	27 (33)	228·2 (270·0)	12·5 (15·5)
Bangladesh, India	46·6 (47·4)	9 (13)	193·1 (274·3)	13·6 (17·0)
Africa	30·9 (30·6)	10 (6)	323·6 (201·3)	13·0 (16·6)
West Indies	16·6 (20·8)	6 (12)	361·4 (576·9)	13·0 (16·1)
Malta, Gibraltar, Cyprus	8·7 (9·3)	– (1)	– (107·5)	8·1 (12·6)
Remainder of New Commonwealth	15·6 (14·1)	2 (1)	128·2 (67·1)	9·9 (9·5)
Pakistan	40·7 (40·2)	5 (8)	122·9 (199·0)	19·1 (22·1)
Other Foreign	56·5 (58·1)	3 (9)	53·1 (154·9)	8·7 (10·8)
Not Stated	0·6 (1·1)	– (4)	– (3,636·4)	107.7 (100.0)

Figures exclude Fortuitous maternal deaths.

* Stillbirths and deaths under 1 week per 1,000 total births.

Source: "Mortality Statistics, perinatal and infant: social and biological factors", Series DH3, nos 14, 15 & 17.

number of women from the ethnic minorities in the younger childbearing age who were born in the United Kingdom.

It is interesting to compare the percentage distribution of total births by birthplace of mother with similar figures for maternal death by their birthplace. From Table 1.9 it can be seen that over 17 per cent of maternal deaths were of women born outside the United Kingdom. Over half of these deaths were of women born in Bangladesh, India and Africa, whereas less than a third of total births to women born outside the United Kingdom were to women born in these countries.

In 1982–1984 the ratio of maternal deaths to total births where the woman was born outside the UK was 42 per cent higher than the ratio for women born in the UK; where the woman was born in the New Commonwealth the ratio was more than twice as great. Similarly the perinatal mortality rate was higher for mothers born outside the UK. The numbers of maternal deaths are small and these are subject to random variation. Therefore care must be exercised in the use of these statistics. However, similar patterns were seen in comparable data for 1979–81.

Age and parity

Maternal mortality is closely related to both maternal age and parity, as shown in Table 1.10. Over the period 1976–1984 it can be seen that at ages 20 years and over maternal death rates rose with age, particularly for mothers aged 35 years and over. There was a similar effect with parity although the age-specific mortality rates for primiparous mothers were generally higher than those associated with the second and third pregnancy.

Table 1.10 *Mortality rates of Direct maternal deaths per million maternities by age and parity, 1976–1984*

Age (years)	Parity					
	1	2	3	4	5+	Total
under 20	90·5	36·8	–	–	–	82·3
20–24	74·3	34·2	46·9	64·4	99·9	57·6
25–29	113·6	61·0	81·6	112·3	40·0	85·7
30–34	114·3	86·9	107·2	117·7	189·3	107·1
35–39	248·9	204·4	236·0	338·6	261·4	252·5
40+	761·3	85·2	704·3	769·4	812·6	653·4
Total	98·9	63·0	105·0	161·0	231·2	95·9

Underlined rates based on fewer than five deaths

The pattern of fertility in terms of age and parity has changed over recent years and those changes can make an important contribution to maternal mortality. Table 1.11 shows the changes in age and parity distributions of maternities over every third triennium between 1955–1957 and 1982–1984. In particular there has been a decreasing proportion of women who have five or more births. Since these women have higher maternal mortality rates than lower parity women, this decrease contributes to the reduction in maternal mortality.

Table 1.11 *Percentage distribution of all maternities by birth order* and age, and by age at first birth, for every previous third triennium*

	1955–57	1964–66	1973–75	1982–84
Birth order				
1	38	36	40	41
2	29	30	36	35
3	15	17	14	16
4	8	8	5	6
5 and over	9	9	4	4
Total	100	100	100	100
Age (years)				
Under 20	5	10	11	9
20–24	29	32	32	30
25–29	32	30	37	34
30–34	20	17	14	19
35–39	10	8	5	6
40 and over	3	3	1	1
Total	100	100	100	100
Age (years) at first birth				
Under 20	12	21	21	18
20–24	45	46	39	38
25–29	28	23	31	30
30–34	11	7	7	11
35 and over	4	3	2	3
Total	100	100	100	100

Source: Unpublished OPCS fertility tables.
* Note: 'Birth order' is defined as the number of live-born children, including the present deliveries, adjusted to convert the number of live births to estimated maternities.

Marital Status

Table 1.12 shows the marital status of the women who suffered a Direct or Indirect maternal death in the triennium covered by this report, with over one fifth of the mothers being unmarried at the time of death. This pattern is consistent with the general trend in the number of illegitimate births; by 1984, 17 per cent of live births were illegitimate. Statistics do not identify unmarried women who were in stable unions.

Data on social class are usually based on the occupation of the woman's partner. These data have not been included in this Report because of the increasing difficulty in interpreting social class information on women, many of whom are working and not influenced by their partner's occupation.

The characteristics and trends for each of the causes of maternal death will be considered separately in the following chapters.

Table 1.12 *Maternal deaths in relation to marital status, 1982–1984*

Marital status	Direct deaths	Indirect deaths	Total	Percentage of series
Married	111	54	165	78·9
Single	16	11	27	12·9
Widowed	2	–	2	1·0
Divorced	5	2	7	3·3
Separated	4	4	8	3·8
Not stated	–	–	–	–
Total	138	71	209	100·0

2. Hypertensive diseases of pregnancy

Summary

There was a welcome reduction in deaths from hypertensive diseases of pregnancy to 25 in 1982–84, after the disappointingly high 36 in 1979–81. However, the number of cerebrovascular deaths actually increased and the proportion with substandard care showed no diminution. It is difficult to interpret the trend in maternal mortality from a single cause over a few triennia and the significance of the improvement in 1982–84 will probably only be clarified by the results in future triennia.

Attention has been drawn to the developments in the management of pre-eclampsia with proteinuria, and the need for expert advice to be available.

In the last report it was stressed that since severe hypertensive diseases of pregnancy are now relatively rare, it can be difficult for every unit to provide the high standard of care required for such cases at all times when individual obstetricians and midwives may have little practical experience of their management. It was therefore suggested that specialist teams should be established in each region to collaborate with other obstetric units by providing advice or taking over cases, if requested. This has not been implemented because some regions believed that the proposal implies that all severe hypertensive diseases of pregnancy should be treated in one centre. This was never the intention, but the increase in cerebrovascular deaths and the unchanging level of substandard care demonstrate the continuing need for special expertise to be available in each region. It is therefore recommended that regional advisory subcommittees in obstetrics and gynaecology review their requirements and propose the organisational improvements they need to their health authorities.

Table 2.1 *Number of women who died from hypertensive diseases of pregnancy and the death rate per million maternities, 1970–84**

Triennium	Total		Pre-eclampsia		Eclampsia	
	Number	Rate	Number	Rate	Number	Rate
1970–72*	43	18·7	14	6·1	29	12·6
1973–75*	34	17·7	15	7·8	19	9·9
1976–78	29	16·6	16	9·1	13	7·4
1979–81	36	18·7	16	8·3	20	10·4
1982–84	25	13·3	11	5·8	14	7·4

*For changes in numbers and rates since last report see page 153.

Pre-eclampsia and eclampsia

In the 1982–84 triennium there were 25 deaths from pre-eclampsia and eclampsia (ICD 642). This compares with 36 in 1979–81, 29 in 1976–78, 34 in 1973–75 and 43 in 1970–72. Eclampsia caused 14 deaths in 1982–84 compared with 20 in 1979–81, 13 in 1976–78, 19 in 1973–75 and 29 in 1970–72. All but one of the known deaths from pre-eclampsia and eclampsia in 1982–84 were the subject of confidential enquiry. The unreported death was certified as due to eclampsia but is not included in the figures.

The number of deaths from eclampsia and pre-eclampsia in 1982–84 is the lowest recorded in the Confidential Enquiries. There were 11 deaths from pre-eclampsia and 14 deaths from eclampsia. While the reduction in deaths from hypertensive diseases of pregnancy in 1982–84 is welcome, the extent to which it is due to chance will be clarified only by the trends in future triennia.

Hypertensive diseases of pregnancy were present in 16 deaths directly attributable to another cause. These have been excluded from this chapter and are counted in the following chapters.

Pulmonary embolism	(Chapter 4)	4
Cerebral Thrombosis	(Chapter 9)	2
Anaesthesia	(Chapter 13)	2
Haemorrhage	(Chapter 3)	1
Amniotic fluid embolism	(Chapter 6)	1
Abortion	(Chapter 11)	1
Indirect deaths:		
Cardiac disease	(Chapter 10)	1
Phaeochromocytoma	(Chapter 14)	4

Four of the five patients who died of pulmonary embolism are counted in Chapter 4 and one in Chapter 11, it is noteworthy that 4 had had abdominal deliveries (3 Caesarean sections and 1 hysterotomy) for severe pre-eclampsia. The fifth delivered spontaneously following intrauterine death at the 37th week of a pregnancy complicated by essential hyptertension. This was the only death associated with essential hypertension in this triennium. Obstetricians should be aware of an increased risk of pulmonary embolism in women with fulminating pre-eclampsia who require abdominal operative delivery.

Died undelivered

Four women died undelivered. One with severe pre-eclampsia developed liver necrosis and died of intra-abdominal haemorrhage at 27 weeks gestation. The other three died of cerebral haemorrhage, one at 34 weeks with typical post-mortem findings of pre-eclampsia, one apparently following eclampsia at 35 weeks with disseminated intravascular coagulation and one at 36 weeks, soon after admission to hospital following fits at home.

Duration of pregnancy at delivery

In the remaining 21 patients, the duration of pregnancy at the time of delivery was:

Duration of pregnancy in weeks at time of delivery	*Number of women who died after delivery*		
	Pre-eclampsia	Eclampsia	All
Up to 28	1	2	3
29–32	1	6	7
33–37	5	4	9
38–40	2	0	2
41+	0	0	0
All	9	12	21

Death from eclampsia occurred earlier in pregnancy than death from pre-eclampsia. Of the 12 eclampsia deaths, 8 had occurred by the 32nd week and only 4 at 33 or later weeks. In the 9 pre-eclampsia deaths, the comparable figures were 2 by 32 weeks and 7 at 33 or later weeks. To include the women who died undelivered would change these proportions very little.

Eleven of the 25 deaths occurred by the 32nd week, including 8 of the 12 from eclampsia. The duration of pregnancy at the time of death therefore has implications for the organisation of antenatal care which are discussed in the conclusions.

Perinatal mortality

Of the 21 mothers who were delivered, two delivered dead fetuses following pregnancy terminations at 26 weeks. In the remaining 19, four delivered stillborn and 15 liveborn infants, one of whom died in the first week. Another died a "sudden infant death", six weeks later. Perinatal mortality was therefore 263 per thousand (5 of 19) compared with 333 in 1979–81, 462 in 1976–78 and 465 in 1973–75

Immediate cause of death

Eclampsia—Autopsy was performed in all of the 14 women dying from eclampsia and the immediate causes of death were reported as follows:

Cerebral haemorrhage	7 cases
Cerebral oedema	3 cases
Cerebral cortical necrosis	1 case
Cerebral softening with small pontine haemorrhage	1 case
Pulmonary oedema (intravenous fluid overload)	2 cases

No death was primarily attributed to hepatic failure, necrosis or haemorrhage, although autopsy revealed characteristic histological changes in the liver in five of these cases.

Pre-eclampsia—Autopsy was performed in 10 of the 11 women dying from pre-eclampsia and the immediate causes of death were reported as follows:

Cerebral haemorrhage (without arterio-venous malformation)	6 cases
Cerebral infarction	2 cases
Cerebral oedema	1 case
Hepatic necrosis (with intra-abdominal haemorrhage)	1 case

Although there was only one death from hepatic necrosis, characteristic liver damage was also found in 3 of the 9 deaths attributed to cerebral complications. Evidence of glomerulonephritis was also found in the woman who died from cerebral oedema. Autopsy was not performed in one woman where death was associated with respiratory difficulty apparently resulting from pulmonary oedema following infusion of blood and crystalloid solutions after operative delivery.

The most important causes of death in women with hypertensive diseases of pregnancy were cerebral haemorrhage, oedema or infarction, present in 21 of 25 deaths. Only one death was attributed primarily to hepatic necrosis but characteristic liver damage was found in another eight cases. Overloading of the circulation with intravenous fluids led to three deaths from pulmonary oedema. Disseminated intravascular coagulation was detected in three of the eclamptic deaths and in one of the pre-eclamptic deaths. No death was directly attributed to the disorder but it undoubtedly contributed to several.

Of the 21 women who delivered, 15 required operative delivery, 10 by Caesarean section, 3 by forceps and 2 by breech delivery.

Age and parity

In Tables 2.2 and 2.3 are shown the actual numbers of deaths by maternal age and parity in comparison with the expected numbers based on the findings over the last four triennia. The fact that the expected number of deaths is greater than the actual number accords with an appreciable reduction in maternal mortality from hypertensive diseases in 1982–84.

Table 2.2 *Number of maternal deaths from hypertensive diseases by age, 1982–84 compared with 1970–81*

Age (years)	1970–81*			1982–84		
	Total maternities	No of deaths	Rate per million	Expected deaths	Total maternities	Actual deaths
Under 25	3,375,015	55	16·3	12	738,436	7
25–29	2,734,413	45	16·5	11	640,524	8
30–34	1,274,954	20	15·7	6	361,923	8
35–39	405,741	17	41·9	5	122,318	2
40 and over	102,222	5	48·9	1	20,552	–
All ages	7,892,345	142		35 (Total by addition)	1,883,753	25

*For changes in numbers and rates since last report see page 153

Table 2.3 *Number of maternal deaths from hypertensive diseases by parity*, 1982–84 compared with 1970–81*

Parity	1970–81**			1982–84		
	Total maternities	No of deaths	Rate per million	Expected deaths	Total maternities	Actual deaths
1	3,143,097	91	29·0	22	762,961	14
2	2,769,818	24	8·7	6	653,502	7
3	1,189,470	14	11·8	3	292,666	3
4	456,430	6	13·1	1	107,223	–
5+	333,530	7	21·0	1	67,402	1
All	7,892,345	142	–	33 (Total by addition)	1,883,754	25

* For definition of parity see page 154
**For changes in numbers and rates since last report see page 153

Number of eclamptic fits

In 13 of the 14 patients who had eclampsia, fits were witnessed by either a doctor or a midwife. Fits occurred antepartum in 8, intrapartum in one, postpartum in 3 and both ante and postpartum in 2. Fits occurred for the first time at home in 6 cases, in a hospital based general practitioner unit in one case and in a consultant unit in 7 cases.

Number of fits before death	Number of women
1	7
2	2
3	1
4	1
'Many'	3

Death following only one or two fits in 9 of the 14 women illustrates the need for early and effective treatment. Of the 14 who died from eclampsia, 7 had been admitted to a consultant unit before having any fits.

Clinical features

Despite atypical clinical features, three deaths were attributed to hypertensive disease of pregnancy because the autopsy findings were characteristic. One young primigravida was delivered spontaneously at 32 weeks of a live, growth-retarded infant. Although well and normotensive at 14 and 28 weeks, within 24 hours of delivery she developed mild hypertension, upper abdominal pain, vomiting and severe leucocytosis, collapsed after eight hours and died in the intensive care unit. Autopsy revealed widespread haemorrhagic hepatic necrosis and a large recent cerebral haemorrhage. The appearances were consistent with pre-eclampsia.

In another young woman in her second pregnancy, the blood pressure did not exceed 130/85 mm of mercury up to 31 weeks. At 34 weeks, she developed headache, vomiting and diarrhoea. Her husband visited the general practitioner who prescribed treatment, but on his return home he found his wife dead, undelivered. Autopsy revealed cerebral haemorrhage and the classical liver damage with periportal vascular lake formation, fibrin deposition and focal necrosis. The spiral arterioles in the placental bed showed no adaptation to pregnancy. Death was attributed to pre-eclampsia.

The third patient, a young primigravida, also died undelivered. She was normotensive throughout pregnancy but at 36 weeks called her practitioner at night because of vomiting, upper abdominal pain and tenderness. He found that she was normotensive, administered metoclopramide and planned a further home visit. Five hours later, she had to be admitted to hospital after repeated convulsions and the blood pressure was 150/100 mm of mercury. There were signs of cerebral haemorrhage and she died a few days later. Autopsy confirmed a massive cerebral haemorrhage, hepatic congestion but no necrosis. Death was attributed to eclampsia.

Care was not considered substandard in any of these three cases. Each illustrates the potentially lethal significance of upper abdominal pain and vomiting in late pregnancy even in normotensive or only mildly hypertensive women.

Pathology

All 24 deaths were counted as Direct. All those due to eclampsia and all but one due to pre-eclampsia came to autopsy. Four of the 23 autopsies were considered anatomically substandard, and thirteen (55 per cent) had inadequate or unrecorded histology to confirm an anatomical diagnosis of hypertensive disease of pregnancy (see chapter 17). It is of interest that the kidneys from two of these patients were removed for possible transplantation.

Substandard care

Care was considered substandard in 18 of the 25 deaths directly caused by hypertensive diseases of pregnancy (72 per cent). Of the 18 deaths, 12 were in women with eclampsia and 6 with pre-eclampsia.

Responsibility for substandard care was often shared. In all, it involved the patient in 3 instances, the general practitioner alone in 5, the practitioner and the midwife in one and the consultant obstetric team in 16. In only one case was anaesthetic care substandard, although death was attributed to anaesthesia in 2 other women with hypertensive diseases of pregnancy who have been counted in Chapter 13. There were no administrative deficiencies.

As in the last report, self-neglect by patients included concealing the pregnancy, refusing to have the blood pressure measured and leaving hospital against medical advice.

Substandard care by a general practitioner alone in five instances and once also with the midwife, mainly involved failure to recognise dangerous symptoms or signs. In these six women, who later died from eclampsia, the warning of the serious condition of the patient provided by epigastric pain and vomiting associated with hypertension, was overlooked in two. In the other four, significant proteinuria was disregarded, probably because the blood pressure was only marginally elevated, although other symptoms or signs were present in three, including headache, blurring of vision or increasing oedema.

Care by the consultant obstetric team was considered substandard in 16 deaths of which 11 were due to eclampsia. Care was considered substandard either because of failure of diagnosis or management.

Failure of Diagnosis Obstetric assessment was considered deficient in two women with proteinuria who were not further investigated; one in whom the combination of epigastric pain, vomiting, hypertension, proteinuria and oedema was attributed to 'acute anxiety' and one in whom there was delay in informing senior staff that postpartum eclampsia had occurred.

While disseminated intravascular coagulation (DIC) was not the attributed cause of death in any case, it led to severe bleeding which contributed to death from cerebral infarction in one pre-eclamptic patient and to pulmonary oedema following over transfusion in another. DIC developed in 6 of the 25 deaths and failure of the consultant team to anticipate it was considered to constitute substandard care in three.

Although hepatic necrosis was the cause of death in only one pre-eclamptic patient liver damage was observed at autopsy in 8 deaths (5 from eclampsia and 3 from pre-eclampsia). In these, liver damage was not detected during life because liver enzymes had not been measured.

Failure of Management In six patients who died from cerebral haemorrhage there was failure to control increasing hypertension. In one woman no antihypertensive treatment was given despite the blood pressure rising to 170/120 mm of mercury during a 9-day antenatal stay in hospital, and in one other woman no antihypertensive treatment was administered for severe postpartum hypertension. Four women died following inadequate blood pressure control, with methyldopa and hydralazine in one, intravenous labetolol and diazepam in the second and intravenous hydralazine in the third and fourth.

Anticonvulsant therapy was inadequate in only one eclamptic death in which diazepam was not administered until after a second fit. Fits occurred or recurred in the 14 eclamptic deaths despite administration of hydralazine in 9, diazepam in 6, chlormethiazole in 4, omnopon or morphine in 4, and labetolol, phenytoin or mannitol, each in 1. Of these drugs, only diazepam, chlormethiazole and phenytoin can be considered anticonvulsant. Lack of treatment was not regarded as substandard medical care in two untreated women since one had concealed her pregnancy and the other died undelivered at home.

Failure to deliver the patient expeditiously enough in pregnancy or labour, or to control worsening hypertension, constituted substandard care in 5 of the 9 'cerebral' deaths in pre-eclampsia and 8 of the 12 'cerebral' deaths in eclampsia. Although the total number of deaths from hypertensive diseases of pregnancy was lower in 1982–84 than in 1979–81 the number of 'cerebral' deaths increased from 17 to 21.

Overload of the circulation by intravenous fluids led to death from pulmonary oedema in one pre-eclamptic and two eclamptic patients. Since the volume of the vascular bed is so much reduced in severe pre-eclampsia and eclampsia and susceptibility to pulmonary oedema is increased, the need for expert control of intravenous fluid administration in such cases is again stressed. Excessive sedation contributed to death from respiratory arrest in three other patients.

Conclusions

In this triennium the number of deaths from hypertensive diseases of pregnancy fell to 25 from 36 in 1979–81. Eleven women with hypertensive diseases died from other direct causes in 1982–84, death being attributed to haemorrhage in one, pulmonary embolism in five, amniotic fluid embolism in one, cerebral thrombosis in two and anaesthesia in two. Since the hypertensive disease usually made a significant contribution to death, it might be thought that the true contribution of hypertensive diseases to maternal mortality had been under-estimated in 1982–84. In 1979–81, however, as well as the 36 deaths directly attributed to hypertensive diseases of pregnancy, another 15 associated with hypertensive diseases were attributed to another, direct cause. The total number of Direct deaths attributed to, or associated with, hypertensive diseases of pregnancy therefore fell from 51 in 1979–81 to 36 in 1982–84.

Care was substandard in 72 per cent of deaths in 1982–84. In several pregnant women the epigastric pain and vomiting which warn of impending eclampsia were disregarded while others who developed significant proteinuria and hypertension were not immediately admitted for investigation.

It is clear that not every obstetric unit maintains its expertise in the management of severe hypertensive disease of pregnancy since 7 of 14 women who died of eclampsia had their first fit after admission to a consultant unit. Cerebral complications were by far the commonest cause of death in this triennium implying ineffective control of blood pressure. The proportion of deaths from 'cerebral' causes in this triennium is one of the highest in the whole series of reports.

It is not known if the improved results in 1982–84 were due to better management than in previous years since the Confidential Enquiries provide no information about non-fatal cases of pre-eclampsia or eclampsia. The examples of substandard care described demonstrate that further improvement can be achieved.

The high incidence of cerebral deaths indicates the need for more effective control of hypertension. The renal, hepatic, or blood coagulation complications of pre-eclampsia can also contribute to death for they can arise insidiously, be more severe or progress more rapidly than might be expected from the level of hypertension alone, whether or not antihypertensive drugs are being administered.

It is deserving of comment that maternal death from eclampsia is commonest before the 32nd week of gestation. This probably results at least in part from women having fewer screening visits at this relatively early stage of pregnancy and from the longer unobserved intervals during which troubles can develop unnoticed. It could be concluded that it is justified for all primigravidae to be routinely screened at least every two weeks from the 24th week onwards. However, since each additional antenatal examination represents a great increase in work, the possibility should first be considered of conducting a trial large enough to show reliably whether or not increased screening from 24 weeks would reduce eclampsia before 32 weeks. The combination of hypertension detected for the first time and significant proteinuria (1+ on routine testing) should lead to immediate hospital admission. If there are also symptoms such as headache, visual disturbances, epigastric pain or vomiting, the condition should be considered as an acute emergency. If the patient is not in hospital, this would justify a call to the flying squad and admitting her to a centre in which suitable intensive care could be provided both for her and her baby.

The routine management of proteinuric pre-eclampsia should include repeated measurements of renal function and of 24 hour protein excretion, platelet counts and plasma liver enzymes. These tests are best done in conjunction with the department of clinical biochemistry. Effective control of blood pressure is mandatory and unless it can be kept consistently below 170/110 mm of mercury, pregnancy should be ended by delivering the baby. A falling platelet count (below $110 \times 10^9/1$), increasing plasma liver enzyme activity or the development of symptoms of impending eclampsia are generally regarded as indications for immediate delivery, possibly by Caesarean section. Continuation of pregnancy with proteinuric pre-eclampsia beyond 36 weeks is rarely justified.

Pre-eclampsia is a complex and variable disorder with many atypical presentations. As cases of life-threatening severity are now rare obstetricians and midwives are likely to have relatively little experience in their management. In the 1979–81 report we recommended the establishment of regional teams with special expertise. The incidence of substandard care in 1982–84 shows the continuing need for such expert advice and clinical assistance to be readily available. Although in several regions high risk pregnancy management groups already exist, which also deal with or advise about severe hypertensive disease in pregnancy, our assessors report that a common response to the suggestion has been that obstetric units are too dispersed for all severe cases to be dealt with in one regional centre. Such complete centralisation was not recommended, however, the proposal being simply that there should be in each region a group with particular interests and expertise in the management of pregnancy at high risk because of hypertensive disease. Such a group should be able to take over cases on request to provide both the intensive obstetric care needed for women with severe early hypertension and the expert neonatal care necessary for the babies who often have to be delivered extremely pre-term.

Regional advisory subcommittees in obstetrics and gynaecology, in conjunction with their regional assessors, should therefore consider the arrangements which would be most suitable for them, and make agreed recommendations to their regional authorities.

3. Haemorrhage

Summary

In 1982–84 there were nine Direct deaths from haemorrhage, the lowest ever recorded in any triennium. Four of these were due to antepartum haemorrhage, three to postpartum haemorrhage and two to haemorrhage due to other obstetrical trauma. The mortality rate fell to 4.8 per million maternities. In 1979–81 by comparison the number of Direct deaths was 14 and the mortality rate 7.3 per million maternities. In 1982–84 there was also some diminution in the proportion of deaths in which care was regarded as substandard, to 6 of the 9 deaths, compared with 12 of the 14 deaths in 1979–81.

Excessive bleeding remains a significant factor in the causation of maternal mortality. In addition to the nine Direct deaths from haemorrhage there were 15 from other causes which were also complicated by haemorrhage and which have been considered in detail in their respective chapters. Haemorrhage also contributed to several of the 10 deaths from ectopic pregnancy. It is again recommended therefore that every obstetric unit should have an agreed procedure for the management of massive haemorrhage. Suggested guidelines for drawing up such a procedure are listed in the annexe to this Chapter.

Haemorrhage

In 1982–84 there were nine Direct deaths from haemorrhage, the lowest ever recorded in these reports. The mortality rate fell to 4.8 per million maternities. There were no late deaths. In 1979–81 14 deaths were directly attributable to haemorrhage and the mortality rate was 7.3 per million maternities. In 1982–84, care was substandard in 6 of the 9 deaths, compared with 12 of 14 deaths in 1979–81.

The nine deaths directly due to haemorrhage in 1982–84 have been classified as follows:

ICD 641	Antepartum haemorrhage	4
ICD 666	Postpartum haemorrhage	3
ICD 665	Other obstetrical trauma	2

Table 3.1 shows the number of Direct deaths from haemorrhage by cause and the death rate per million maternities in the five triennia from 1970–72 to 1982–84.

Deaths from blood loss related to Caesarean section for antepartum haemorrhage are included in this chapter but as in previous reports, deaths from

Table 3.1 *Number of deaths from haemorrhage and rates per million maternities 1970–84**

	1970–72*	1973–75	1976–78	1979–81	1982–84
Placental abruption	6	6	6	2	2
Placenta praevia	6	2	2	3	2
Postpartum haemorrhage	18	13	16	9	3
Other obstetrical trauma	–	–	–	–	2
Total	30	21	24	14	9
Rate per million maternities	13·1	10·9	13·7	7·3	4·8

*For changes in numbers and rates since last report see page 153.

Table 3.2 *Actual compared with expected number of women who died from haemorrhage by age, 1982–84 compared with 1970–81**

Age (years)	1970–81*			1982–84			
	Total maternities	Number of deaths	Rate per million	Expected deaths	Total maternities	Actual deaths	Rate per million
Under 25	3,375,015	24	7·1	5	738,436	–	–
25–29	2,734,413	20	7·3	5	640,524	3	4·7
30–34	1,274,954	22	17·3	6	361,923	2	5·5
35–39	405,741	12	29·6	4	122,318	3	24·5
40 and over	102,222	11	107·6	2	20,552	1	48·7
All ages	7,892,345	89	–	22 (Total by addition)	1,883,753	9	4·8

*For changes in numbers and rates since last report see page 153.

Table 3.3 *Actual compared with expected number of women who died from haemorrhage by parity*, 1982–84 compared with 1970–81***

Parity	1970–81**			1982–84			
	Total maternities	Number of deaths	Rate per million	Expected deaths	Total maternities	Actual deaths	Rate per million
1	3,143,097	28	8.9	7	762,961	1	1.3
2	2,769,818	16	5.8	4	653,502	6	9.2
3	1,189,470	13	10.9	3	292,666	1	3.4
4	456,430	13	28.5	3	107,223	–	–
5+	333,530	19	57.0	4	67,402	1	14.8
Total	7,892,345	89	–	21 (Total by addition)	1,883,753	9	4.8

*For definition of parity see page 154.
**For changes in numbers and rates since last report see page 153.

haemorrhage attributed to abortion, ectopic pregnancy and ruptured uterus are discussed in detail in their relevant chapters.

The nine deaths directly attributed to haemorrhage are discussed in this chapter. The 15 deaths attributed to other causes are considered in their relevant chapters. Antepartum haemorrhage complicated 4 of the 15 deaths which were directly attributed to hypertensive diseases of pregnancy with cerebral haemorrhage in one (Chapter 2), pulmonary embolism in two (Chapter 4) and amniotic fluid embolism in one (Chapter 6). Postpartum haemorrhage complicated the other eight deaths which were attributed to ruptured uterus in two (Chapter 8), miscellaneous causes in one (Chapter 9), cardiac disease in three (Chapter 10), anaesthesia in one (Chapter 13) and infusion of infected blood in one, which was counted as an Indirect death (Chapter 14). In Chapter 17 attention is also drawn to the rare but dangerous complication of infected blood being transfused. In three cases haemorrhage complicated abortion (Chapter 11).

Table 3.2 shows that the actual number of deaths from haemorrhage in 1982–84 was lower than expected, the risk of death increasing with age as in previous triennia (1970–81). The death rate by age per million maternities from 1970–1984 was as follows:

Age in years	<25	25–29	30–34	35–39	40+	All years
Death rate per million maternities	5.8	6.8	14.7	28.4	97.7	10.0

Table 3.3 shows that the large fall in deaths from haemorrhage in 1982–84 occurred in all parity groups except parity 2, when compared with previous triennia (1970–81). Over the whole period (1970–84) however, the death rate from haemorrhage remained higher in primigravidae than in women of parity 2. The death rate by parity per million maternities for 1970–84 was as follows:

Parity	1	2	3	4	5+	All
Death rate per million maternities	7.4	6.4	9.4	23.1	49.9	10.0

Placental abruption

Two deaths were caused by placental abruption. In both, severe blood coagulation defects developed. The first patient was 40 years of age and was persistently hypertensive throughout her 6th pregnancy. She refused admission to hospital earlier in pregnancy but agreed to be admitted for induction of labour three days before term. Shortly after admission and before induction was attempted she suffered an antepartum haemorrhage of 500 ml and blood transfusion was commenced. Coagulation was normal. Labour progressed rapidly and the patient was delivered spontaneously of a live infant. After delivery, bleeding persisted from the mother's nose and mouth as well as from the vagina and four units of blood were transfused. Within 80 minutes of delivery there was evidence of defibrination. Shortly afterwards the patient had a fit and died. At autopsy, there was no evidence of hypertensive disease, amniotic fluid embolism or uterine rupture, but the placenta was not available for examination.

The second patient was admitted with clinical signs of placental abruption and intrauterine fetal death at the 35th week of her second pregnancy. Blood clotting was normal. Blood transfusion was commenced, morphine administered and labour augmented with intravenous oxytocin in increasing dosage. Just before spontaneous delivery of a stillborn infant, the mother's blood pressure fell to 90/50 mm Hg and after delivery of the placenta with adherent blood clot, there was continuing vaginal haemorrhage and a progressive coagulation defect developed. Following administration of fresh blood and fresh frozen plasma, there was a transient, incomplete recovery and then the patient collapsed again, the haemoglobin level falling to 5.8 g/dl. There was no evidence that the fall in haemoglobin concentration had been due to overtransfusion with crystalloid solutions. She died 22 hours later in the ICU. Autopsy was not performed.

In both cases placental abruption has been accepted as the most reasonable diagnosis.

Placenta praevia

Two deaths were associated with placenta praevia. A low lying placenta had previously been revealed by ultrasound during pregnancy, leading to delivery by emergency Caesarean section.

The first patient was admitted to hospital at 32 weeks in her pregnancy after an ultrasound scan had revealed placenta praevia. After a month she took her own discharge but was readmitted two days later because of antepartum haemorrhage. Caesarean section soon afterwards confirmed the diagnosis of placenta praevia. The operative blood loss was 1000 ml and two units of blood were transfused. Two hours later disseminated intravascular coagulation accompanied by torrential vaginal bleeding occurred. Although hysterectomy was performed by the consultant, death occurred within three hours. At autopsy the pathologist found no evidence of amniotic fluid embolism. The excised uterus was apparently not examined.

The second patient was admitted at the 32nd week of pregnancy with antepartum haemorrhage and an ultrasound scan found placenta praevia. She was managed conservatively in hospital but after five weeks there was further haemorrhage and emergency Caesarean section was performed. Pre-operatively, the blood pressure was normal and haemoglobin level 12.5 g/dl. The operation progressed uneventfully until, during suture of the uterine wound, the blood was noted to be dark blue. Generalised bleeding developed, thought most likely to be due to a coagulation defect. Despite blood transfusion and active resuscitation, the patient's condition deteriorated rapidly and she died about one hour later. Autopsy confirmed the presence of coagulation failure but revealed no evidence of amniotic fluid embolism. No explanation was found for the extremely rapid onset of coagulation failure and collapse. It was suggested that the blood loss might have been underestimated, and the possibility that collapse was due to the infusion of infected blood was not excluded since the blood remaining in the infusion set was not cultured. Anaesthetic factors could not be entirely excluded.

Postpartum haemorrhage

Three deaths were directly caused by postpartum haemorrhage. The first was of a type to which these reports have repeatedly drawn attention. The patient had had two Caesarean sections. At operation the placenta was anterior, partially praevia and accreta but was successfully removed without excessive bleeding. Within two hours, however, torrential vaginal haemorrhage and hypovolaemic shock developed. Total hysterectomy was performed. Bleeding was severe and persisted from vessels in the lateral pelvic wall despite removal of the left tube and ovary. There was no coagulation defect. Despite further laparotomy, ligation of the internal iliac vessels and re-suturing of the vaginal vault, bleeding persisted and the patient died 10 days later of adult respiratory distress syndrome, despite excellent intensive care.

In the second woman, who had had one previous Caesarean section, operative delivery was followed by secondary haemorrhage from the abdominal wall and the development of a coagulation defect. Despite appropriate treatment and two further abdominal wall explorations to try to control this bleeding she developed renal failure and brain death occurred nine days after delivery.

In the third patient, labour was induced at 42 weeks and a live infant delivered by forceps. There was a postpartum haemorrhage of one litre but despite transfusion of two units of blood the blood pressure remained at 80/60 mm of mercury. A vulval haematoma was discovered and evacuated under general anaesthesia, three further units of blood being transfused. A coagulation defect was suspected because the cavity of the evacuated haematoma showed general oozing rather than a single bleeding point, but there was no other evidence to support this diagnosis. In the recovery phase she became dyspnoeic and cyanosed and pulmonary oedema was suspected. Despite treatment with intravenous frusemide administration and ventilation she died two hours later. No obvious explanation for death was found at autopsy. There was no pulmonary oedema, and although initial histological examination suggested amniotic fluid embolism this was not confirmed on review.

'Other obstetrical trauma'

Two other deaths which resulted from haemorrhage after delivery have been described separately from the three postpartum haemorrhage deaths because the bleeding was not from the uterus, cervix or vagina.

In the first patient, the placenta was retained following spontaneous delivery, labour having been induced at 38 weeks by vaginal prostaglandin administration because of hypertensive disease. Manual removal was delayed for over two hours because the obstetric staff had to perform two emergency Caesarean sections in other patients. Despite the delay, bleeding was minimal and her condition remained excellent. When manual removal was performed under general anaesthesia the uterus was found to be bicornuate and the placenta firmly adherent. The procedure was nevertheless uncomplicated and the blood loss 300 ml. Anaesthesia could not be reversed, and within 5 minutes the peripheral pulses were undetectable. Ten minutes later the patient was dead. At autopsy, there had been an intra-abdominal haemorrhage of 3.5 litres. The uterus was intact and the source of bleeding was said to be tears in the 'right flank mesenteries' in the peritoneum near the caecum where there was also retroperitoneal haemorrhage. Uncertainty remains as to exactly which vessels were torn. Death undoubtedly resulted from massive intra-abdominal haemorrhage in the absence of uterine damage.

In the second woman, a multipara, following spontaneous face-to-pubes delivery the placenta was delivered normally by controlled traction, with a blood loss of only 200 ml. About 90 minutes later, however, the systolic blood pressure had fallen to 70 mm Hg and a mass was palpable anterior to the uterus in the left lower abdomen just below the umbilicus and apparently arising from the pelvis. A large paravaginal, retroperitoneal haematoma was suspected and blood transfusion was commenced. Within 40 minutes the abdomen had been opened. One litre of extraperitoneal blood clot was evacuated. There was no blood in the peritoneal cavity and the source of the haematoma appeared to be large vessels in the region of the bladder neck and paravaginal area. At first, bleeding seemed to be controlled by sutures and by pressure on a pack in the area. The abdomen was closed with drainage of the retropubic space. A urinary catheter and large vaginal pack were inserted. Before long, however, further profuse bleeding necessitated a second exploration at which both internal iliac arteries were ligated and other small vessels clipped. Bleeding appeared to be from a surgically inaccessible site. In all, 36 units of blood were transfused before the patient died, 44 hours after delivery. Although there was a transient episode of hypocoagulability, failure of coagulation was not the main cause of the continued bleeding. This latterly required one unit of blood every two hours to maintain central venous pressure but despite this , the haemoglobin level dropped from 10 to 7 g/dl. Ultimately, irreversible ventricular fibrillation developed. Autopsy revealed an intact uterus flanked by an extensive, diffuse, extra-peritoneal haematoma arising from the left side of the pelvis which extended up the anterior abdominal wall, and laterally and posteriorly, up to the level of the ribs. A fresh, oval, 8 cm haematoma was found within the levator ani muscle in the left paravaginal area and no source for the bleeding could be identified.

In neither of these unusual and very diffcult cases was care substandard.

Coagulation failure

Abnormalities of coagulation were detected or suspected in six of the nine deaths from haemorrhage. Coagulation failure predisposed to the bleeding which persisted after delivery in both deaths from placental abruption. It was also suspected in both the deaths attributed to placenta praevia although the diagnosis was based entirely on clinical observation of spilled blood not clotting in one and of 'bleeding occurring from several sites' in the other. It was present in only one of the three deaths from postpartum haemorrhage and in neither of the deaths from "other obstetric trauma". There was thrombocytopenia in the woman who bled from a haematoma in the levator ani muscle but it was transient, occurred only at a late stage and was not the main cause of bleeding. In no case was there delay in detecting development of a coagulation defect. Disorders of coagulation are also discussed in Chapter 17.

Pathology

All of the nine deaths were counted as Direct. Eight came to autopsy. Only one of the eight autopsies was judged to be anatomically substandard, but histology was inadequate or unrecorded in three; in two of these no proper search had been made for evidence of disseminated intravascular coagulation although this diagnosis was suggested in the anatomical reports. Failure to obtain an autopsy in the ninth death was regrettable since the clinical diagnosis of placental abruption had to be accepted without confirmation, and uterine rupture and amniotic fluid embolism could not be excluded.

Substandard care

Care was regarded as substandard in six of the nine deaths from haemorrhage in 1982–84 and included self-neglect by two patients, one discharging herself against advice and the other refusing admission, as well as involving the anaesthetic team in one case mentioned in Chapter 13. In the three remaining deaths, substandard care by the consultant obstetric team involved either delay with treatment or other unsatisfactory management. In one death, the consultant team did not perform hysterectomy or internal iliac artery ligation which might have been life saving, and in the other, did not perform surgery early enough.

In one case, the care was considered substandard because the uterus removed for severe post partum haemorrhage following Caesarean section for placenta praevia was not sent for examination.

Conclusions

Review of the deaths from haemorrhage in 1982–84 reveals no obvious recurring problems. Attention is again drawn to the special risk of placenta accreta associated with anterior placenta praevia in women previously delivered by lower segment Caesarean section, especially more than one previous section.[1] Although previous reports have stressed the dangers of delegating elective Caesarean section to inexperienced obstetricians, especially section for anterior placenta praevia, the patient with placenta praevia accreta is at risk of severe haemorrhage even with an experienced operator. In a woman previously delivered by Caesarean section and in whom elective Caesarean section is planned, the placental site should be identified by ultrasound before operation. If the placenta is anterior and praevia, the increased risks are high enough to merit special preparations, including cross matching more blood than usual (4–6 units), alerting the anaesthetist and theatre staff, discussing the possible hazards with the patient and, if possible, her partner and ensuring adequate intravenous infusion sites from the start of the procedure.

In the 1979–81 report 12 of the 14 cases of substandard care involved the obstetric team and included failure to deal efficiently with severe haemorrhage and coagulation failure. In 1982–84 by contrast, the consultant obstetric team was only involved in 3 of the 6 cases of substandard care mainly involving delay in considering hysterectomy or internal iliac artery ligation. There were no instances of ineffective management of coagulation failure.

To consider only the 9 deaths directly due to haemorrhage would be to underestimate the influence of excessive bleeding on maternal mortality. Haemorrhage contributed to another 15 deaths which were directly attributed to other causes. It therefore seems useful to review here some specific instances in 1982–84 in which haemorrhage contributed to maternal deaths attributed directly to other causes.

Disseminated intravascular coagulation or liver necrosis led to severe bleeding in some women with proteinuric hypertensive diseases of pregnancy. The need to anticipate such complications by regular platelet counts and liver enzyme assays has again been emphasised (Chapter 2).

Administration of anticoagulants in women who require, or have already had, a surgical procedure. Serious bleeding has occurred in women on anticoagulants on whom surgery was performed without special precautions, or when anticoagulants were administered to women following recent surgery apparently without appreciation of the risk of haemorrhage or the need for careful clinical and haematological surveillance (Chapter 4, 10 and 11).

Undue susceptibility to the effects of haemorrhage or fluid overload has been a factor in the deaths of some women with severe heart disease, especially of congenital type (Chapter 10).

Failure to recognise the signs of hypovolaemia in intra-abdominal bleeding or from an extra-pelvic source has been a feature of several deaths. The possiblity of internal haemorrhage always has to be kept in mind as a cause of collapse in women of reproductive age or following evacuation of a pregnant uterus (Chapters 5 and 11).

Transfusion of infected blood is an extremely rare cause of collapse but obstetric units should continue to ensure that a remnant of each unit of blood given is preserved for a time agreed with their own blood transfusion service to make possible subsequent bacteriological investigation, if necessary. (Chapter 14).

Suggested guidelines for the management of massive obstetric haemorrhage have again been listed in the Annexe to this chapter. Regional assessors have reported concern by obstetricians that a consultant team might be criticised unfairly, if in their unit the advice in the annexe was not followed to the letter. We stress that the annexe simply provides *guidelines* based on the advice we have received. Its aim is to assist obstetric teams draw up protocols appropriate to their own units.

Reference

1. Clarke S L, Koonings P and Phelan J P. Placenta praevia/accreta and prior Caesarean section. *Obstetrics and Gynaecology* 1985; **66:** 89.

Annexe to Chapter 3

Guidelines for the management of massive obstetric haemorrhage

Summon all the extra staff required, including obstetricians, anaesthetists, midwives and nurses. Alert the haematologists and the blood-transfusion service. Make sure porters are available.

At least two peripheral infusion lines will be necessary. The cannulae used should be not less than 14 gauge. Central venous pressure monitoring should immediately be set up since it helps ensure that therapy is safely controlled. Central venous pressure should be continuously displayed and a display of intra-arterial pressure is also extremely useful.

Facilities for the measurement and display of CVP, intra-arterial pressure, ECG, heart rate, blood gases and acid base status should be available to all consultant obstetric units.

A large sample of blood should be taken (eg 20 ml or an amount agreed by the local departments concerned) for blood grouping, cross matching and investigating coagulation. A minimum of six units of blood should be ordered. Whole blood is the treatment of choice but if only plasma reduced blood is available at first, additional colloid will be necessary if more than three units of plasma-reduced blood have to be given. Human albumin solution (4·5%) is best, but degraded gelatin solutions may be more readily available and are often used in practice. Hetastarch preparations may also be useful.

Dextran interferes with blood coagulation and cross-matching. No more than one litre should ever be given and then only in acute emergency. In fact its use is now regarded as contraindicated in severe bleeding.

All patients should be given blood of their own group as soon as possible. Patients known to have anti-c antibodies from their antenatal records should not be given Group 0 Rh-negative blood. For other patients with severe haemorrhage however, uncross-matched Group 0 Rh-negative blood can be life saving.

Regular haemoglobin or haematocrit assessment can be helpful in the control of blood and fluid therapy, but restoration of normovolaemia is the first priority. Do not give fresh frozen plasma or platelet concentrates until major haemorrhage has been stopped or until approximately 5 units of stored blood have been given rapidly. It is a waste of these scarce materials to give them before this, particularly if there is active bleeding from a source which should be controlled by immediate surgery.

Pressure infusion with a compression cuff on the plastic bag is essential. Martin's pumps and chambers for hand pumping do not give blood fast enough in exsanguinated patients and should not be used.

Blood should be administered through blood warming equipment.

Blood filtration is not only unnecessary, it adds positive dangers because it delays giving blood.

Additional calcium administration is rarely required but if indicated, 10% calcium chloride is preferable to calcium gluconate.

All patients with more than moderate continuing haemorrhage require proper monitoring of pulse rate, blood pressure and CVP, blood gases, acid base status and urinary output as well as dedicated continuing care by the midwifery, nursing and medical staff. Consideration should be given to the potential advantages of transfer to an ICU.

4. Pulmonary embolism

Summary

Pulmonary embolism continues to be one of the major causes of Direct maternal deaths. There were 25 Direct deaths attributed to pulmonary embolism. Seven other deaths are mentioned in this chapter, four of whom died from pulmonary embolism but are counted in other chapters. All 29 deaths are analysed in this Chapter but three other Direct deaths from cerebral sinus thrombosis whose deaths are discussed are not included since they did not die as a consequence of their pulmonary embolus.

Excluding all deaths following abortion and ectopic pregnancy, 9 deaths occurred during pregnancy, 4 in the first trimester, 2 in the second and 3 in the third. Four deaths followed vaginal delivery and 12 deaths followed Caesarean sections. There is a 26-fold greater risk of death from pulmonary embolism after Caesarean section compared with vaginal delivery. Risk factors for pulmonary embolism include Caesarean section, particularly if performed as an emergency, obesity, hypertensive diseases of pregnancy, excessive blood loss and any further major surgery. Age has again been shown to be a more important risk factor than parity.

Pulmonary embolism

There were 25 deaths coded to pulmonary embolism (ICD 673.2). Nine occurred in pregnancy and 16 after delivery. Of the latter, 12 followed Caesarean section and 4 vaginal delivery. There were four other deaths from pulmonary embolism, one after an ectopic pregnancy (ICD 633.1) and three after legal abortion (635.6). One of these occurred after a colectomy was carried out, 8 days after the legal abortion, for ulcerative colitis, one 3 days following a prostaglandin termination and one 28 days after an abdominal hysterotomy for fulminating pre-eclampsia. These 29 deaths have been included in the discussion and tables in this chapter.

Three other women in whom deep vein thrombosis and pulmonary embolism were found at autopsy are reviewed in this chapter because of their interest. All these women died from cerebral (sagittal sinus) venous thrombosis not pulmonary embolism and are counted in Chapter 9.

Deaths from amniotic fluid embolism are considered in Chapter 6.

Table 4.1 compares the number of deaths from pulmonary embolism with previous reports.

Table 4.1 Deaths from pulmonary embolism, 1970–84*

	1970–72*	1973–75	1976–78	1979–81	1982–84	Total
Deaths after abortion or ectopic pregnancy	10	3	2	5	4	24
Deaths during pregnancy	14	14	14	11	9	62
Deaths during labour	–	–	–	1	–	1
Deaths after vaginal delivery	22(11·6)	13(7·7)	20(12·7)	4(2·3)	4(2·4)	63
Deaths after Caesarean section	15(145·2)	6(59·2)	9(74·6)	7(41·9)	12(64·6)	49
Total	61	36	45	28	29	199

Rates per million vaginal deliveries or Caesarean section deliveries given in parentheses.
*For changes in numbers and rates since last report see page 153.

The total number of deaths in 1982–84 was one more than in 1979–81. The ratio of deaths from pulmonary embolism following Caesarean section to deaths following vaginal delivery in 1970–72, 1973–75 and 1976–78 was 12:1, 8:1 and 6:1 respectively, in 1979–81 it increased to just over 16:1 and was even greater in 1982–84 being 26:1. This reflects either a fall in deaths from pulmonary embolism following vaginal delivery in the last two of these four triennia or the increased use of Caesarean section, particularly as an emergency procedure.

Tables 4.2 and 4.3 show the time internal between delivery and fatal pulmonary embolism following vaginal delivery and Caesarean section in the 15 years 1970–1984.

Table 4.2 Interval between delivery and pulmonary embolism following vaginal delivery, 1970–84*

	1970–72*	1973–75	1976–78	1979–81	1982–84	Total (%)
Up to 7 days	9	7	7	3	1	27 (43)
8–14 days	7	–	2	–	2	11 (17)
15–42 days	6	6	11	1**	1	25** (40)
Total	22	13	20	4*	4	63**(100)

* For changes in numbers since last report see page 153.
**Includes one in which the time of birth was concealed by the mother but the interval, based on the autopsy findings, was within 42 days.

Table 4.3 Interval between delivery and pulmonary embolism following Caesarean section, 1970–1984

	1970–72	1973–75	1976–78	1979–81	1982–84	Total (%)
Up to 7 days	5	2	5	3	3	18 (37)
8–14 days	3	2	2	2	3	12 (24)
15–42 days	7	2	2	2	6	19 (39)
Total	15	6	9	7	12	49(100)

It is apparent from Tables 4.2 and 4.3 that deaths from pospartum pulmonary embolism can occur at any time, whatever the mode of delivery, although the most dangerous period appears to be in the first week after delivery. It should be noted that the majority of the fatal episodes occurred after the mother would have been expected to leave hospital.

Pulmonary embolism after abortion and ectopic pregnancy

There were three deaths from pulmonary embolism following legal abortion. In one case, suction curettage was performed as a therapeutic measure for severe ulcerative colitis and total colectomy carried out eight days later was a major factor in this case. The risks of hospitalisation and major surgery shortly after pregnancy are well recognised.

The second case was of an obese woman a grand multipara who had been sterilised ineffectively. She was admitted three months later with a suspected ectopic pregnancy. At laparotomy a normal 14 week pregnancy was found and tubal ligation was carried out. Following the laparotomy, termination of pregnancy was requested. This was carried out six days later by extra-amniotic prostaglandin, with the catheter introduced under general anaesthesia. The following day the patient aborted and the uterus was evacuated under another general anaesthetic. On the first post-operative day pain in one leg was thought to be musculoskeletal. A venogram revealed an extensive thrombus of the deep veins of the calf and anticoagulant therapy was commenced. However, on the second post-operative day she collapsed and died from a large pulmonary embolism confirmed at autopsy.

The third case was that of a young primigravida who required an emergency abdominal hysterotomy for fulminating pre-eclampsia which did not respond to intensive therapy. The fetus was stillborn and there was a retroplacental haematoma and a placental chorioangioma. She was discharged home on the 5th day but re-admitted on the 16th day with symptoms suggestive of pulmonary embolism. There was no evidence of deep vein thrombosis. Despite appropriate heparin therapy her condition deteriorated and pulmonary artery embolectomy was carried out when she was moribund. At thoracotomy however, little embolic material was obtained. Her condition did not improve and she died the next day. Autopsy revealed embolic material in the distal parts of the pulmonary artery in both lungs. Recent thrombus was found in the left femoral vein and its branches but did not correspond in age or histological appearance with the embolic material in the pulmonary arteries. This was not, therefore, a straightforward case of massive pulmonary embolism. Although it is possible that the initial chest symptoms were due to emboli, the fatal episode appeared to be due to extensive primary pulmonary artery thrombosis which occurred despite anticoagulant therapy.

In the last report it was noted that there had been only one death from pulmonary embolism in association with the 150 deaths from ectopic pregnancy recorded since they were first reported in 1964–66. The second such death occurred in 1983. This woman, a heavy smoker, had deep vein thrombosis confirmed by a venogram and a possible pulmonary embolism in 1978 when on oral contraceptives. At 6–7 weeks gestation in this pregnancy she was admitted with a threatened abortion and an ultrasonic scan suggested an intrauterine pregnancy and a right ovarian cyst. A further scan was arranged and she was allowed home to rest but was admitted as an emergency the following day. The diagnosis of ruptured ectopic pregnancy was made and a right salpingectomy performed. Although a history of probable deep vein thrombosis was obtained no details were available. Active post operative mobilisation was encouraged and she was discharged on the 7th post-operative day only to die from a massive pulmonary embolism 38 days after the salpingectomy. Autopsy revealed extensive thrombosis in both calf veins. Unfortunately no information was available about whether any warning symptoms had been present or if combined oral contraceptives had been recommenced.

Pulmonary embolism during pregnancy

There were 9 deaths from pulmonary embolism in pregnancy, 4 in the first trimester, 2 in the second and 3 after the third.

Only one woman had had anticoagulant therapy. She was admitted in the 28th week of her second pregnancy with symptoms suggestive of pulmonary embolism. She was treated with anticoagulants for a few days but this was discontinued after review and she was discharged. She continued to feel unwell and was breathless when seen daily by the midwife. On the 5th day after her discharge she was re-admitted moribund and suffered terminal cardiac arrest. Autopsy confirmed a pulmonary embolus originating in the pelvic veins.

The second patient, a visitor to the UK, who had had a deep vein thrombosis after her second confinement five years previously, was seen at seven weeks gestation. Three weeks later she was brought in dead. Forty-eight hours earlier she had complained to her husband of pain in one leg but they did not seek medical advice. Autopsy revealed a large pulmonary embolism but no evidence of pelvic, femoral or calf vein thrombosis.

The third case was that of a tall, slim 17-year-old girl who was found dead in bed. Autopsy revealed a pulmonary embolus, pelvic vein thrombosis and an unsuspected 6–7 week pregnancy. Although she was known to have started oral contraceptives 11 months before her death it was not established that she had continued taking them. It is of interest that she had attended her GP with a cough three days prior to death but there was no clinical reason at that time to suspect pulmonary embolism.

Another woman at 12 weeks gestation was admitted under the care of a consultant physician because of chest pain. A chest radiograph taken was consistent with an infection or infarction and although the possibility of pulmonary embolism was raised she was allowed home five days later. She was readmitted a few hours after discharge in a semi-comatose state and had a cardiac arrest from which she did not recover. Autopsy revealed a large pulmonary embolus with thrombi in both calves and in the pelvic veins.

A diabetic patient on antituberculosis therapy had a legal abortion. Four months later she developed neck stiffness and was admitted to a neurology unit. A Computed Tomography (CT) head scan revealed a non-specific abnormality on the right side but no diagnosis was made. Her condition began to deteriorate and continued to the stage of brain death. She was certified dead four days after admission. An incidental finding at autopsy was an unsuspected 12 weeks pregnancy. Large emboli were found in both pulmonary arteries and the right common iliac vein was occluded by thrombus. There was also thrombosis of the sagittal sinus with extension into the cerebral veins. An area of fibrosis with caseation was found in the apex of the right lung.

Four women died suddenly and unexpectedly, in pregnancy, of pulmonary embolism. There were features in all cases which should have alerted the attendants to the possibility of thromboembolism. These included obesity, breathlessness, inactivity and immobilisation, and a previous history of thromboembolism. The source of embolism was confirmed at autopsy as being in pelvic veins in two instances and the calf veins in one. Autopsy was not performed on one woman who had had a thoracotomy to remove an embolus after initial cardiac arrest.

Pulmonary embolism following vaginal delivery

Four deaths occurred after normal vaginal delivery. In two instances there were warning symptoms but the diagnosis was missed and in the two other cases heparin was started too late.

One or more risk factors for the occurrence of thromboembolism were present in each case. These included obesity or excessive weight gain in pregnancy, postpartum sterilisation, high parity, immobilisation and in one case the early use of oral contraceptives commenced by the woman herself. All four women were aged under 35 years. The diagnosis was confirmed by autopsy in three women. Thrombus was found in the pelvic veins of two but not in the third, despite a careful search. The diagnosis was accepted on clinical grounds in the remaining case, because an inadequately treated pulmonary embolism was followed by the typical features of a massive and fatal pulmonary embolus.

Pulmonary embolism after Caesarean section

There were 12 deaths from pulmonary embolism within 42 days of delivery by Caesarean section, 2 in 1982, 3 in 1983 and 7 in 1984. The operation was elective in one case and carried out as an emergency during labour in 11 cases because of fetal distress or failure to progress in labour. Apart from the risk factor of Caesarean section itself all the women had other characteristic features associated with an increased risk of venous thrombosis and/or embolism. Details of these are as follows:

Number of risk factors present in the 12 women who died of pulmonary embolism after Caesarean section:

Obesity or excessive weight gain	5 women
Further major surgery required. Blood transfusion in excess of 2 units. Hypertensive disease of pregnancy. Warning deep vein thrombosis Age 35 years and over. Para 3 and over.	Each factor present in 3 women
Previous history of thromboembolism Immobilisation after discharge. Haemorrhage.	Each factor present in 2 women.
Development of paralytic ileus and warning pulmonary embolism, respectively	Each factor present in one woman.

Autopsy was carried out on 10 of the 12 patients who died of pulmonary embolism following delivery by Caesarean section. Thrombus was found in the pelvic veins in 4 cases, in the pelvic and leg veins in 3 cases and in the leg veins in one case. Despite a thorough search, no site of thrombus was found in two cases. In the two women who had no autopsy, the diagnosis had been confirmed by pulmonary angiogram in one, and the other had a proven deep vein thrombosis which was being treated with prophylactic subcutaneous heparin. She collapsed and died with the characteristic signs of a massive pulmonary embolus.

Cerebral venous thrombosis and thromboembolism

There were three Direct deaths due to cerebral sinus thrombosis (ICD 671.5) counted in Chapter 9. All three autopsies disclosed the presence of a pulmonary embolus and evidence of pelvic or calf vein thrombosis.

The first woman, a multipara, who had severe agoraphobia which made care difficult, had a fit on the 15th postpartum day after a normal term delivery. A history of weakness in the right arm seven days previously and two grand mal attacks was obtained. On admission to a medical unit her blood pressure was 210/105 mm Hg and clinical signs of right sided motor neurone involvement were present. After some initial improvement, her condition deteriorated on the 6th day after admission. She developed bilateral upper motor neurone signs in the legs, became unconscious and died the following day, ie 24 days after her delivery. Autopsy revealed that all the major cortical sinuses contained thrombi confirming the clinical diagnosis which had been made, but in addition there was a deep vein thrombosis in the right calf and a right sided pulmonary embolus.

The second woman was an overweight young primigravida who developed mild pre-eclampsia and delivered spontaneously almost at term. On the 22nd day of an uneventful puerperium she was admitted to a neurosurgical unit because of generalised headache. She developed a right hemiparesis, became comatose, and died on the 24th day after delivery. Autopsy revealed that death was due to cortical vein and venous sinus thrombosis. There was also thrombus in the left common iliac vein, numerous pulmonary emboli in the segmental arteries of the right lung, and a large embolus in the pulmonary artery of the lower lobe.

The remaining case, an elderly parous patient, had an elective Caesarean section at 38 weeks because of hypertension and the presence of fibroids. A suspected deep vein thrombosis on the 6th day was treated with support stockings and she was discharged home on the 10th day. She was readmitted on the 20th post-operative day with a definite deep vein thrombosis which was treated with anticoagulants. Ten days later she had cerebral signs and a mild left hemiparesis. She was transferred to a neurosurgical unit, since a cerebral haemorrhage was suspected. Twelve hours later, after several fits and confirmation of a large cerebral haematoma, a craniotomy was carried out and the haematoma evacuated. There was minimal improvement and death occurred 12 hours later, 32 days after delivery. The autopsy was of poor quality but it did reveal a sagittal sinus thrombosis, pulmonary embolus and thrombosis of the left femoral vein.

Factors associated with death from pulmonary embolism

The aetiology of thromboembolism is *multifactorial.*

Age and parity

Table 4.4 shows the maternal deaths due to pulmonary embolism by age and parity and the estimated rates per million maternities for 1970–1984.

The risk of fatal pulmonary embolism increases sharply with advancing age particularly after 35 years and is more significant than increasing parity.

Obesity

In this report, 10 out of 29 women who died were reported to be obese and 5 of the 12 patients who died after Caesarean section were overweight.

Hypertension, restricted activity and mode of delivery

Several of the women had been admitted for rest during the pregnancy because of hypertension, with or without oedema. Some were relatively immobile in the puerperium. Four of the five overweight women who required delivery by Caesarean section were also hypertensive and had been in-patients prior to delivery.

Table 4.4 Maternal deaths due to pulmonary embolism by age and parity and estimated rates per million maternities, 1970–84***

Age (years)		Parity**					
		1	2	3	4	5+	All
Under 20	Number	10	–	–	–	–	10
	Rate	12·8	–	–	–	–	10·4
20–24	Number	17	12	4	1	–	34*
	Rate	10·9	10·4	12·2	11·3	–	10·8
25–29	Number	23	25	10	7	2	67
	Rate	20·1	18·3	17·8	35·0	19·7	19·9
30–34	Number	9	8	9	4	7	37
	Rate	26·7	13·7	21·6	23·3	54·1	22·6
35–39	Number	1	4	10	9	9	33
	Rate	13·8	29·9	74·4	104·3	89·2	62·5
40 and over	Number	1	1	2	5	8	17
	Rate	80·7	45·1	85·5	263·6	174·4	138·5
All ages	Number	61*	50	35	26	26	198*
	Rate	15·6	14·6	23·6	45·9	65·4	20·3

* Excludes one with parity not stated.
** For definition of parity see page 154.
*** For changes in numbers and rates since last report see page 153.

Suppression of lactation by oestrogens

None of the women who died after delivery were reported to have had lactation suppressed by oestrogens.

Previous thromboembolism

In three women it was recorded that thromboembolism had occurred previously. All were on combined oral contraceptives when the thrombosis occurred.

Pathology

All 29 deaths discussed in this chapter were considered as Direct. Twenty-five were coded to pulmonary embolism and 21 of these came to autopsy. Two of the four women on whom no autopsy was performed had had an embolectomy which confirmed the diagnosis. Of those who died, coded to other causes, all but one came to autopsy.

Seven of the autopsies were technically substandard, either because a search for the site of origin had not been made or recorded, or because essential histology had not been reported.

Substandard care

As in previous reports, failure to administer anticoagulants has not been considered to be substandard care. It is often difficult to diagnose thromboembolism during pregnancy and the puerperium, and failure to do so has not been regarded as representing substandard care.

Women at high risk of thromboembolism often have obstetrical complications requiring prolonged bed-rest or operative delivery. In such cases, all acceptable methods of avoiding stasis in the pelvic and leg veins should be considered, including ambulation, physiotherapy and anti-thrombosis stockings. If anticoagulant therapy is commenced in the puerperium it should be continued for at least four to six weeks after all the symptoms and signs have disappeared and should be controlled by proper monitoring of the state of coagulation of the blood.

Discussion

Unexpected deaths from pulmonary embolism still occur and in some of the reported cases, either the past history or the symptoms or the signs should have alerted the attendants. Full awareness of the possibility of thrombosis and embolism is essential if the number of deaths is to be reduced, since pregnancy, labour and the puerperium are associated with venous stasis in the pelvic and leg veins and there is an increased clotting tendency which favours the risk of thrombosis.

The clinical features of venous thrombosis are less specific in pregnant women because of the greater involvement of the pelvic veins and the physiological changes in pregnancy and the puerperium. The detection of reduced blood flow in the femoral vein by non-invasive methods can be helpful and a positive result is an indication for anticoagulant therapy. If the patient is considered to have a pelvic vein thrombosis she should be either treated with anticoagulants or have a diagnostic venogram, with appropriate shielding of the fetus, to visualise the external and common iliac veins. Treatment is then determined according to the result.

The clinical diagnosis of a major pulmonary embolus is not difficult, but small emboli are often missed or ignored. Chest X-rays and ECGs can be normal in the presence of pulmonary emboli so that they do not exclude the diagnosis. For a proper diagnosis, a perfusion lung scan with or without a ventilation scan, or pulmonary angiography might be necessary. Perfusion and ventilation scanning expose the fetus to minimal irradiation and the risks of any of these investigations have to be weighed against the risks of not treating pulmonary embolism.

When there is any doubt about whether the diagnosis is embolism or infection it is safer to treat the patient with anticoagulants *and* antibiotics until the correct diagnosis is established. It is unlikely that the administration of heparin will prejudice the outcome of the antibiotic treatment of infection and the risk of giving anticoagulants with proper control, particularly heparin, in the short term is not high and tends to be over estimated.

Long-term sequelae such as pulmonary hypertension are possible after recovery from thromboembolism, particularly pulmonary embolism. Anticoagulant therapy is often required for several months after the initial deep vein thrombosis or pulmonary embolism to minimise their sequelae. The potential risks of the long-term anticoagulant therapy chosen should be known and therapy should be properly monitored.

Analysis of the risk of death from pulmonary embolus during pregnancy or the puerperium reveals that it is increased in women who are aged over 35 years, of high parity, overweight, have restricted activity, a Caesarean section, or any operation subsequent to delivery. It should be appreciated that women with severe pre-eclampsia or hypertension are a high risk group because they are often overweight, usually in hospital for a considerable time and therefore relatively immobile, and because they are often delivered by Caesarean section in the interests of their baby or themselves. In this triennium, 5 of the 29 women who died from pulmonary embolism had severe pre-eclampsia and another had hypertension, that is almost 20 per cent of the deaths. A history of deep-vein thrombosis or pulmonary embolism in previous pregnancies always warrants serious consideration of prophylactic anticoagulant therapy. If the diagnosis of thromembolism has been confirmed it helps determine the management of any future pregnancy.

It should also be remembered that puerperal women can die from thrombosis in the cerebral venous system with or without evidence of thrombosis elsewhere. It seems likely that the improved standard of the autopsies now carried out will continue to reveal, as in this Enquiry, that thrombosis, when present, is more widespread than had been realised previously.

5. Ectopic pregnancy

Summary

There were 10 Direct deaths, and 3 other deaths in women with ectopic pregnancies from anaesthetic complications. This is the lowest number of deaths from this cause in any triennium. The death rate from ectopic pregnancy per million estimated pregnancies halved, falling from 7·9 in 1979–81 to 4·0 in 1982–84. The fall in mortality occurred despite an increase in the incidence of ectopic pregnancy.

The primary site of implantation of the conceptus, with the exception of the cervical pregnancy, was in the fallopian tube, but in one instance after rupture, it had secondarily implanted on the broad ligament. Three women died suddenly at home, one of whom, most unusually, died a few weeks after a hysterectomy, from an unsuspected tubal pregnancy.

The changing methods and importance of diagnosing and managing ectopic pregnancy are discussed. In three cases the failure to consider the diagnosis and carry out a vaginal examination in early pregnancy was regarded as substandard care.

Non-caucasian women were again found to be at increased risk.

Ectopic pregnancy

In this triennium, 14 women had an ectopic pregnancy and died, but only 10, including one who had a pulmonary embolism, have been counted as Direct deaths due to ectopic pregnancy. Anaesthesia was the cause of death in three cases and one Indirect death due to myocardial infarction is counted in Chapter 10. All 13 Direct deaths are discussed in this chapter.

Of the 10 Direct deaths due to ectopic pregnancy, the conceptus was in the fallopian tube in nine (ICD 633.1) but in the other instance, it had, after rupture of the fallopian tube, implanted on the broad ligament (ICD 633.8). In the three deaths attributed to anaesthesia, the conceptus was in the fallopian tube, and it was in the cervix of the woman counted in Chapter 10 who died from a myocardial infarction as the pregnancy was being spontaneously aborted.

Since the 1976–78 triennium all known cases of death from ectopic pregnancy have been reported.

Ectopic pregnancy, prior to this triennium, had become one of the four principal causes of death. The marked fall in deaths from ectopic pregnancy in 1982–84 could be a chance finding. Yet, since the Hospital In-Patient Enquiry (HIPE) figures indicate that the incidence of non-fatal cases increased (Table 1.7), so the improvement possibly reflects earlier diagnosis and treatment.

There will always be difficulty in estimating the number of women at risk from ectopic pregnancy. However, the number of deaths from ectopic pregnancy has been compared in Table 5.1 with the estimated number of pregnancies as defined in Chapter 1. Although there was little change in the death rate between the figures for the three previous triennia, a definite reduction occurred this time.

Eleven of the 13 Direct deaths were in the first trimester which is similar to the last report.

Table 5.1 Deaths from ectopic pregnancies and rates per million estimated pregnancies

Triennia	Total estimated pregnancies*	Ectopic pregnancies* in HIPE	Ectopic pregnancies per 10,000 estimated pregnancies	Number of deaths	Death rates per million estimated pregnancies	Death rates per 1000 ectopic pregnancies
1970–72	2890·7	11·6	40	34	11·8	2·9
1973–75	2578·4	11·7	45	19	7·4	1·6
1976–78	2323·0	11·6	50	21	9·0	1·8
1979–81	2543·2	12·1	48	20	7·9	1·7
1982–84	2507·0	14·4	57	10**	4·0	0·7

* In thousands

** There were 3 other deaths from anaesthesia.

Racial status

Four of the 14 women (28·6%) were born outside the United Kingdom and were of West Indian or Asian origin which as in previous years confirms a greater risk for these women.

Clinical features

Anaesthetic difficulties were encountered on four occasions during the management of ectopic pregnancy. In three instances anaesthesia was the direct cause of death and these cases are discussed in Chapter 13. In each of these three cases there were doubts about the clinical diagnosis. The first woman had been admitted to a general surgical ward for observation and the diagnosis of ectopic pregnancy was made only during laparotomy for suspected appendicitis. The second woman was initially thought to have a threatened and later an incomplete abortion but examination under anaesthesia revealed a pelvic mass. A laparotomy and left salpingectomy was then performed. The third woman had a history of bleeding and pain for 15 days which was considered to be due to pelvic inflammatory disease since a pregnancy test was negative. It was then decided that there were retained products of conception in the uterus and a curettage was arranged for 10 days later. Two days before her planned admission she had acute pain and bleeding following coitus. She was admitted as an emergency for an examination under anaesthesia, followed by laparoscopy and laparotomy, at which cardiac arrest occurred.

The fourth woman who had previously had an ectopic pregnancy was admitted to an Accident and Emergency (A and E) Department in a shocked and semicomatose state. Early laparotomy and clamping of the fallopian tube was done but there was failure to control the hypovolaemia. There was also difficulty with endotracheal intubation. This case is mentioned in Chapter 13, but counted in this chapter.

Two women died at home. One had complained of severe abdominal pain in the early hours of the morning and had to be helped to the toilet by her husband. Her pain improved but she was found several hours later, dead in bed. The other woman, alone at home and in obvious distress, had managed to make a 999 call but was found dead by the ambulance men.

One woman attended the hospital because of abdominal pains but no abnormality was detected. She was considered to have had an intrauterine pregnancy, confirmed by ultrasonic scan, and was booked for hospital confinement. The following day she collapsed and was brought in, dead, to the A and E Department. Autopsy revealed a rupture of the uterus with the fetus in the abdomen but no report of the site of the rupture was made. It seems likely that the pregnancy was cornual. The ultrasonic scan report undoubtedly misled the junior doctor who had seen her on the preceding day.

There were three cases in which a vaginal examination might have established the diagnosis but it was omitted. The first woman attended a gynaecological unit with abdominal pain. A pregnancy test was positive, she was assumed to have a threatened abortion and was admitted for observation. Two days later the possibility of an ectopic pregnancy was raised as a result of a vaginal examination. The diagnosis was dismissed following a further examination. There was no further blood loss and apart from some lower abdominal tenderness she appeared to be well. The following day she was allowed home without a further examination, to be reviewed the following week because she requested a legal abortion. Shortly after arriving home she collapsed, was seen by her GP, but was dead before the ambulance arrived. Autopsy confirmed a ruptured tubal pregnancy.

The second woman collapsed and died within a few minutes of being brought into the A and E Department. She had been seen by four general practitioners on eight separate occasions in the preceding 26 days because of suspected bowel disease. Early in the series of visits, her haemoglobin was found to be 7·8 g/dl but no action was taken and no vaginal examination ever carried out. Autopsy revealed that there had been episodes of bleeding with formation of omental adhesions to the large bowel and a recent rupture of the right fallopian tube with an ectopic gestation adherent to the left broad ligament.

The third woman, who had had a previous first trimester abortion had symptoms attributed to gastroenteritis, despite the presence of vaginal bleeding and acute abdominal pain early in her second pregnancy. The general practitioner who visited her at home was aware of previous pelvic inflammatory disease but avoided a vaginal examination because he felt that it might jeopardize the pregnancy. The patient died later the same day. Autopsy revealed two litres of fresh blood and 600 ml of blood clots in the pelvis from a ruptured right tubal pregnancy.

A very obese woman with an irregular menstrual cycle had lower abdominal pain for four days prior to being admitted to a surgical unit in severe hypovolaemic shock. After resuscitation and review by a gynaecologist, an ectopic pregnancy was diagnosed. An immediate laparotomy was performed at which three litres of blood were removed from the abdominal cavity and a left salpingectomy carried out. Forty-eight hours later she developed symptoms suggestive of pulmonary embolism and was given anticoagulants but a few hours later she had a cardiac arrest from which she did not recover. No evidence of pulmonary embolism or thrombosis was found at a substandard autopsy.

The woman who died from pulmonary embolism had been admitted with pain thought to be due to a threatened abortion. An ultrasonic scan was reported to reveal an intrauterine pregnancy and ovarian cyst. She was sent home to rest but was readmitted the following day to the A and E Department. The diagnosis of ectopic pregnancy was obvious and a laparotomy performed. The fatal embolism occurred 38 days later.

One woman had an unrecognised tubal pregnancy at the time of a hysterectomy operation. She collapsed at home 5 to 6 weeks later and died. Autopsy revealed a ruptured tubal pregnancy.

Pathology

All 10 deaths were counted as Direct, nine of which came to autopsy. Three autopsies were regarded as technically substandard including one in which a uterine rupture was said to be present although no details were given. Five contained no histological report on the fallopian tubes.

Substandard care

Ectopic pregnancy is always a possibility in any woman of reproductive age, who complains of abdominal pain and/or vaginal bleeding. Several cases illustrate the failure of medical attendants to appreciate this. Even experienced observers can find it difficult to be certain that an early pregnancy is within the uterus. A high index of suspicion about the probability of ectopic pregnancy is essential.

When a diagnosis of threatened abortion is made it is always wise to consider the possibility of an extrauterine pregnancy. There is no good evidence that abortion of an intrauterine pregnancy is more likely to occur after a gentle vaginal examination. In three of the fatal cases, failure to perform a vaginal or rectal examination contributed to the adverse outcome.

Discussion

Although the death rate from ectopic pregnancy has markedly decreased in this triennium, the incidence of the condition has increased. Many factors are likely to have contributed to this increase, including a higher incidence of pelvic inflammatory disease associated with greater sexual freedom and modern methods of contraception. The increase in tubal surgery, including the development of microsurgical techniques and the increased use of sterilisation and its reversal, has also contributed. To some extent, the increase in ectopic pregnancies could result from fewer being missed, with laparoscopy being used more and more in cases of doubt. Sophisticated ultrasound examination and the rapid and sensitive assays of the beta sub-unit of hCG (human chorionic gonadotrophin) have probably also detected ectopic pregnancies which in the past might have resolved before becoming a clinical problem.

All health personnel likely to be consulted by women of reproductive age need to be aware of the advances in the methods for diagnosing and managing ectopic pregnancy if they are to provide optimal care. Ectopic pregnancy is of particular importance to women who desire further pregnancies, for conservative tubal surgery is now often possible. Whilst it generally falls to the gynaecologist to manage cases, all doctors providing the primary consultation, including the general practitioner, the casualty or admitting officer, or nursing colleagues to whom responsibility has been delegated, should be aware of the principles involved in early diagnosis.

Even the very sensitive beta-hCG pregnancy test has its limitations. A positive test does not necessarily differentiate ectopic pregnancy from an intrauterine one, although the level tends to be lower in ectopic than in intrauterine pregnancy. In doubtful cases, serial estimations can help establish the diagnosis. Real-time ultrasound or sector scanning alone rarely detect ectopic pregnancy itself but can confirm an intrauterine pregnancy. Although vaginal ultrasound probes provide clear imaging and more reliable diagnostic accuracy, they are not yet widely available and experience with them is limited. At present, a positive test for beta hCG in serum or urine, coupled with an absence of a pregnancy sac in the uterus or elsewhere in the pelvis on an ultrasonic scan, is highly suggestive of ectopic pregnancy. This combination of findings indicates the necessity for laparoscopy or serial hCG estimations depending on the duration of amenorrhoea and whether or not there has been pain, bleeding or pelvic tenderness. Persistent pain requires investigation by laparoscopy.

The need for timely operation for a bleeding ectopic has always been stressed in these reports. Immediate laparotomy with control of haemorrhage is the main life saving measure, and in shocked patients, must take precedence over laparoscopy for diagnostic purposes. Rapid blood transfusion is also vital. By contrast, in fit, normotensive women with unexplained pain, bleeding or pelvic tenderness, laparoscopy plays an important diagnostic role.

Within the whole range of ectopic pregnancy, extratubal ectopic gestation has always accounted for an unduly high proportion of the mortality in these reports. Their late presentation and relative rarity makes cervical and cornual pregnancy difficult to diagnose. The details in this chapter should assist their earlier diagnosis, better management and improved outcome.

6. Amniotic fluid embolism

Summary

In the triennium 1982–84 there were 14 deaths from histologically proven amniotic fluid embolism. There was a fall in the number of deaths per million maternities from this cause from 9.4 in 1979–81 to 7.4 in 1982–84. One woman, who died from haemorrhage as a consequence of a ruptured uterus, also had amniotic fluid embolism, but is counted in Chapter 8. There were two other cases in which a diagnosis of amniotic fluid embolism was initially suspected but after detailed autopsy analysis, the deaths were allocated to other direct causes.

Labour was induced with prostaglandin pessaries in four women and augmented in one instance and because of this association the need for careful monitoring is stressed. Uterine lacerations were found in two women who died from amniotic fluid embolism, one of whom had labour induced with a prostaglandin E_2 pessary.

Only 8 of the 15 infants delivered from the 14 mothers survived the neonatal period.

The management of a woman who survived eight days after amniotic fluid embolism is described.

Amniotic fluid embolism

Since 1973 the diagnosis of death from amniotic fluid embolism has been accepted only if there is positive confirmatory histological evidence. In this triennium there were 14 such deaths classified under ICD 673.1. The death rate per million maternities was 7.4 compared with 9.4 for 1979–81. In addition there was one death due to rupture of the uterus, counted in Chapter 8, in

Table 6.1 *Deaths from amniotic fluid embolism histologically confirmed and deaths suspected of being amniotic fluid embolism but not confirmed, 1970–84**

	1970–72*	1973–75*	1976–78	1979–81	1982–84
Histologically confirmed cases	14(2)	14(1)	11(-)	18(-)	14(1)
Suspected cases	8	7	8	6	2

The numbers in parentheses ( ) are deaths with amniotic fluid embolism present but attributable to other Direct causes.
*For changes in numbers and rates since last report see page 153.

which amniotic fluid embolism was also present. A specific note has been made of the cases in which there were lacerations or tears in the uterine muscle without complete rupture. Two other deaths had features of amniotic fluid embolism. These were clinical in one instance and based on the initial pathological findings in the other. The diagnosis was not, however, confirmed in either case, and the deaths have been allocated to other Direct causes of death.

Clinical features

Two women collapsed and died undelivered at home. One went into spontaneous labour at 35 weeks and the other at 36 weeks gestation. In both cases the diagnosis was confirmed at autopsy and there was no evidence of uterine laceration.

One woman with pre-eclampsia had an intrauterine death at 34 weeks gestation. Labour was induced by intravenous *Syntocinon* followed by a prostaglandin pessary. Because progress was slow two more prostaglandin pessaries were inserted into the vagina. She continued to labour, but died suddenly and unexpectedly about 15 minutes after the spontaneous rupture of the membranes at 5 centimetres dilatation.

Two women, who were in labour at term, collapsed in the second stage. One died as the baby's head was being delivered after the spontaneous onset of labour and spontaneous rupture of the membranes late in labour. The other woman, who had labour induced with a prostaglandin pessary followed by amniotomy, collapsed during the second stage of labour. She was partially resuscitated before delivering her child spontaneously but then had profuse bleeding, collapsed again and died.

Three women who were at term, had emergency Caesarean sections. One woman collapsed in the first stage of labour following the introduction of an intrauterine catheter, another had a presumptive diagnosis of placental abruption and evidence of disseminated intravascular coagulation. Both operations were done because of the mothers' critical condition. The third woman who had labour induced with a prostaglandin pessary developed fetal distress after what appeared to be a convulsion.

One woman went into spontaneous labour at 27 weeks. The membranes ruptured spontaneously during labour and after the delivery of a 750 g girl she was given syntometrine. Shortly afterwards, a second infant was expelled within its membranes and following delivery of the unsuspected second twin an intramuscular injection of ergometrine was given. She collapsed and died from amniotic fluid embolism. An additional finding at autopsy was severe pulmonary hypertension, which had not been diagnosed during life, and might well have been exacerbated by the adminstration of two doses of 0.5 mg of ergometrine.

Three cases presented unusual features. The first woman, a young primigravida, had labour induced by a prostaglandin pessary at 39 weeks because of excessive weight gain. A *Syntocinon* drip was commenced during labour. Shortly afterwards she had what was clinically considered to be an eclamptic fit followed by cardiac arrest and death. Postmortem Caesarean section was carried out and a liveborn infant was delivered. A very detailed autopsy revealed that death resulted from widespread amniotic fluid embolism, without any histological evidence of eclampsia.

The second woman, also a primigravida, suffered from essential hypertension which was controlled during pregnancy by hydralazine and oxyprenolol hydrochloride. Labour began spontaneously at 41 weeks gestation her blood pressure being 160/100 mm Hg. Headache and fever which developed during the first stage were initially ascribed to septicaemia, but later considered to reflect disseminated intravascular coagulation. She died one hour after forceps delivery of a stillborn infant. A detailed autopsy confirmed that death was due to amniotic fluid embolism without any histological evidence of intravascular coagulation.

In an isolated maternity unit, the third woman in her third pregnancy, was delivered by emergency Caesarean section at 33–34 weeks for placenta praevia of a major degree. Shortly after delivery the mother had a cardiac arrest. Following immediate resuscitative measures her condition improved and the operation was completed. The mother was transferred to the ICU of the District General Hospital, five miles away. The next three days were spent correcting a disseminated intravascular coagulation during which time the mother had two laparotomies. At the first, a subtotal hysterectomy was performed but as it did not control the blood loss ligation of both internal iliac arteries was carried out the following day. Her condition improved but over the next 48 hours she developed localised fits and was transferred to a neurosurgical unit, she died, without further treatment, eight days after delivery. Histological appearances in the lungs were consistent with the development of amniotic fluid embolism.

It is considered that the survival of the mother for so long may well have been due to the fact that a high priority was given to the maintenance of oxygenation, support of the cardiac output and blood pressure.

Two women at, or just after term, were found to have lacerations (incomplete rupture) of the uterine wall, thus providing an obvious site for entry of amniotic fluid. One woman had labour induced with a prostaglandin pessary and the other went into labour spontaneously. Both women had considerable blood loss and collapsed in the third stage of labour, one having had a normal delivery and the other a forceps delivery. A third woman, whose death was attributed to haemorrhage following a ruptured uterus but who also had a demonstrable amniotic fluid embolism, is counted in Chapter 8.

Initial symptoms

The initial symptoms considered to be related to amniotic fluid embolism occurred during the:

1st stage	8 cases
2nd stage	3 cases
3rd stage or within one hour	3 cases

This contrasts with the 1979–81 report when one third of the cases presented after delivery.

The outcome for the baby

Eight of the 15 babies survived, 3 died in utero, 2 were stillborn and 2 subsequently died (neonatal twin deaths).

Coagulation disorders

Four of the 14 women developed a coagulation disorder in association with the amniotic fluid embolism. The symptoms and signs of disseminated intravascular coagulation can predominate and obscure the diagnosis of amniotic fluid embolism. This point should be remembered by the pathologist at autopsy.

Induction and augmentation of labour

Labour was induced by means of drugs in 5 of the 14 women. In four women prostaglandin pessaries were used, while one had a *Syntocinon* drip augmented by prostaglandin pessaries on three subsequent occasions. There was insufficient evidence in the reports to assess whether uterine contractions were more powerful than normal in any of these patients. The need for monitoring uterine contractions in labour is discussed in Chapter 8.

Uterine rupture and genital tears

Two of the 14 women counted in this chapter were found to have lacerations involving the uterine wall, and a third woman, who also had amniotic fluid embolism, was considered to have died from a uterine rupture, and is counted in Chapter 8.

Age and parity

The age and parity of the women for all cases since 1970 are given in Table 6.2.

Table 6.2 *All maternal deaths with amniotic fluid embolism present* by age and parity and estimated rates per million maternities, 1970–84*

Age (years)		Parity**					
		1	2	3	4	5+	All
Under 20	Number	2	1	–	–	–	3
	Rate	2·6	6·1	–	–	–	3·1
20–24	Number	6	5	1	2	–	14
	Rate	3·8	4·3	3·0	22·7	–	4·4
25–29	Number	7	6	3	2	– 18	
	Rate	6·1	4·4	5·3	10·0	–	5·3
30–34	Number	2	5	6	3	2	18
	Rate	5·9	8·6	14·4	17·5	15·4	11·0
35–39	Number	1	2	3	3	6	17
	Rate	13·8	14·9	22·3	34·8	59·5	28·4
40 and over	Number	–	1	1	1	4	7
	Rate	–	45·1	42·8	52·7	87·2	57·0
All ages	Number	18	20	14	11	12	75
	Rate	4·6	5·8	9·5	19·4	30·2	7·7

* See Table 6.1 page 47.
** For definition of parity see page 154.

Pathology

All 14 deaths were counted as Direct and all came to autopsy. No autopsy was anatomically substandard and the histology was satisfactory by definition. In two additional patients, who were initially suspected of having amniotic fluid embolism the diagnosis was not substantiated after careful review of the histology.

Substandard care

In the majority of cases there was insufficient evidence to identify any specific factors related to amniotic fluid embolism, although the clinical management of some cases was questionable.

It is again noteworthy that 5 of the 14 women had prostaglandin pessaries to induce or augment labour. The recommended dose was exceeded in one case when used to augment labour.

Suspected cases of amniotic fluid embolism

There were two cases in which amniotic fluid embolism was suspected but not confirmed. The first death of a woman who had a post-partum haemorrhage and required the evacuation of a vulval haematoma, is described in detail in Chapter 3. There was no coagulation defect but prior to her death the patient developed dyspnoea and peripheral cyanosis which raised the suspicion of amniotic fluid embolism. This was not confirmed at autopsy.

The other death is included in Chapter 9 as one of the sudden deaths of unknown cause in pregnancy. The suspicion in this case was due to the initial report of material of possible fetal origin in a frozen section of lung tissue. Detailed analysis of the haematoxylin and eosin stained preparations failed to confirm the presence of any amniotic fluid in the lungs.

Discussion

There were 14 proven deaths from histologically proven amniotic embolism in this triennium and one other confirmed case which for the purposes of the Enquiry, is counted under ruptured uterus in Chapter 8. Two cases mentioned above were suspected of having amniotic fluid embolism but this was not confirmed. This is the smallest number of doubtful cases and reflects a greater awareness on the part of pathologists of the need for careful histological examination of the lungs of patients who die unexpectedly during labour or immediately after delivery.

The age and parity of the 75 mothers who had amniotic fluid embolism since 1970 were analysed in order to assess if age, parity, or both, influenced the occurrence of this condition. This revealed similar findings to embolism due to blood clot, namely the death rate increased after the age of 35 years and after the third pregnancy.

As in the previous reports, no patient presented with symptoms before the onset of labour, although the information about the two women who died at home was limited. Since only one patient presented within one hour of delivery, no assessment can be made in this report as to whether symptoms are more severe in those who present before or after delivery. The relatively high fetal mortality (53 per cent) described supports the opinion expressed in the last triennium that the risk is greater for babies of mothers whose symptoms appear before delivery.

Four of the 14 women who died had received prostaglandins to induce labour. A fifth who had received *Syntocinon* to induce labour also had prostaglandin augmentation on three occasions. One of these five women was found to have a uterine laceration. The evidence is insufficient to confirm or refute the dangers of prostaglandins or oxytocic drugs in the induction or augmentation of labour. It does, however, emphasise the need for as much intensive care and monitoring of uterine activity when prostaglandin pessaries are adminstered as is given when intravenous *Syntocinon* is used. This is paticularly relevant when both drugs are administered.

One woman survived for eight days possibly because of the success of maintaining oxygenation and positively supporting cardiac output and blood pressure. The treatment of the coagulopathy associated with amniotic fluid embolism is still controversial. The replacment of blood loss with whole blood or fresh frozen plasma and packed cells is widely accepted, but the use of heparin or tranexamic acid is still debatable.

7. Genital sepsis

Summary

In this triennium two deaths occurred from genital sepsis, excluding three deaths coded to abortion. This is the lowest number recorded in a triennium. There was only one case of substandard care, and the woman herself was responsible. The other case involved virulent organisms which failed to respond to standard treatment.

Genital sepsis

There were five deaths due to infection of the genital tract in 1982–84, of which three after spontaneous abortion have been counted in Chapter 11. They have been classified as follows:

Sepsis after spontaneous abortion	ICD 634.0	3
Sepsis after surgical procedures	ICD 639.2	1
Sepsis before or during labour	ICD 658.4	1

Table 7.1 shows that the steady decline in deaths from sepsis from all causes has been maintained with each triennium.

Table 7.1 *Maternal deaths from sepsis including abortion with estimated rates per million maternities*, 1970–84*

	1970–72	1973–75	1976–78	1979–81	1982–84	Total
Sepsis after abortion	36	6	7	7	3	59
Puerperal sepsis	13	8	6	2	0	29
Sepsis after surgical procedures	16	11	9	4	1	41
Sepsis before or during labour	1	–	–	2	1	4
Total	66	25	22	15	5	133
Rate*	28.7	13.0	12.6	7.8	2.7	13.6

Sepsis after abortion

There were three deaths from sepsis after spontaneous abortion and none after legal abortion. The three cases illustrate how rapidly death can occur with a particularly virulent organism despite appropriate treatment.

In the first case, the woman who was 12 weeks pregnant presented to her general practitioner with a 'flu-like' illness. She started to bleed vaginally a few hours later and was transferred to hospital. On admission she had no physical signs of infection and the uterus was evacuated under general anaesthesia. At the end of the operation the patient suddenly collapsed and a consultant gynaecologist decided to proceed to immediate laparotomy. At operation there was no evidence of trauma, but petechiae were seen on the surface of both ovaries with haemorrhage in the broad ligament. A diagnosis of disseminated intravascular coagulation due to acute septicaemia was made. Energetic and appropriate treatment failed to alter the course of her illness and she developed pulmonary oedema, dying some hours later. An autopsy with cultures confirmed a streptococcal septicaemia.

The second case was that of a multiparous woman at 22 weeks gestation admitted to hospital feeling well but with a watery vaginal discharge. A complete abortion followed. Two days later she developed a cough with blood stained sputum and endotoxic shock. Intravenous antibiotics were started immediately and membranes were removed from the uterus under general anaesthesia. She developed renal failure and, died three days later in the renal unit. Permission for an autopsy was refused, but blood culture during life revealed an *E. Coli* infection.

The third case involved a multiparous woman with a 14 week pregnancy. She came to the casualty department of her own accord because she was passing clots and she thought she might be pregnant. On examination the cervix was found to be open. She was observed in hospital but six hours later she suddenly collapsed and, despite all attempts to resuscitate her, died within 20 minutes. An autopsy revealed *Clostridium septicum* septicaemia. This history is suggestive, but not conclusive of, criminal interference.

Sepsis before or during labour

There was one death from sepsis during pregnancy in a young woman who concealed her pregnancy up to the time of death. Four days before she died she returned home from work saying she was feeling unwell and went to bed. On the day of her death her relatives found her collapsed and called the flying squad. Efforts to resuscitate her were initially successful but she had a cardiac arrest in the ambulance and died. An autopsy done immediately after death revealed an otherwise healthy young woman with a uterus containing a fetus weighing 3.0 kg, with the cervix fully dilated and the head impacted in the pelvis. There was evidence of generalised intrauterine inflammation with a positive culture of *bacterioides fragilis*. It seems likely that death was due to infection of the amniotic cavity following obstructed labour, possibly of four days duration. The death would surely have been avoided if she had attended for antenatal care.

Sepsis after surgical procedures

One woman died from septicaemia following a Caesarean section. Her history illustrates the impossibility of preventing death in the presence of a virulent infecting organism despite satisfactory care. She was a young multiparous woman who was delivered at term by an uncomplicated intrapartum Caesarean section. Her condition was satisfactory until 17 hours after delivery when she was found to have bacteraemic shock. Wide spectrum antibiotics and penicillin were given immediately but her condition failed to improve. Despite transfer to an intensive care unit where every facility necessary for her resuscitation was used, she died three days later from a group A haemolytic streptococcal septicaemia which was thought to have originated from the upper respiratory tract.

Puerperal sepsis

No deaths could be directly attributed to puerperal sepsis. There were two indirect deaths. One, of septicaemia superimposed on rheumatic heart disease, is described in Chapter 10. The other, a case of septicaemia, developed in a woman with longstanding hypoplastic anaemia. The death is described here because of its relevance to the chapter, but is counted in Chapter 14.

A young woman who had required multiple transfusions for hypoplastic anaemia since she was a child had a planned pregnancy despite the known risks to her life. With the support of regular transfusions to maintain her haemoglobin the pregnancy progressed well until 24 weeks gestational age when petechiae developed on her abdomen and there was bleeding from the nose, lips and gums. She was found to be pancytopaenic with a urinary tract infection and subsequently a diagnosis of septicaemia was made. She was successfully treated with a platelet infusion and antibiotics. Further episodes of septicaemia and bleeding, including haematemesis, were treated at 29,31 and 33 weeks gestation. Labour started at 33 weeks and she had a spontaneous delivery under antibiotic cover with a platelet infusion. She then became pyrexial although blood cultures remained negative until just before she died when *E. coli* was cultured. Obstetrically, all progressed well with involution of the uterus. However, despite a haemoglobin level of 10.0 g/dl, she was severely neutropaenic. Quite suddenly, five days after delivery, she developed profound gram negative shock and despite all efforts to resuscitate her, including large doses of an antibiotic to which the organism was sensitive, she died on the following day. The autopsy did not reveal any specific focus of infection. This case is described in detail because it illustrates the dangers of pregnancy in someone suffering from aplastic anaemia. Despite the understandable desire of this woman and her husband to have a baby, it is our view that this is one of the few medical conditions which becomes lethal in pregnancy. It justifies a recommendation of sterilisation before there is any possibility of pregnancy, or the need for legal abortion for an unplanned pregnancy.

Pathology

All five deaths described in this chapter were considered as Direct. Three have been coded to abortion of which two came to autopsy. Two are coded to genital sepsis. No autopsy was considered to be technically substandard but two of the four (50 per cent) lacked necessary histological confirmation. In all five, adequate microbiological investigations had been made during life, at autopsy, or both.

Substandard care

There were no cases during this triennium in which medical care was substandard. This is in contrast to the previous triennium, 1979–81, when there were several cases in which delay in instituting appropriate treatment was commented on. The only case of substandard care was of a woman who had concealed her pregnancy.

Conclusion

Deaths from genital sepsis have fallen in number once again. It seems likely that this is due to the earlier presentation for medical care of women having a spontaneous abortion, the continued improvement of surgical techniques for legal abortion and the more ready availability of legal abortion. The possibility of overwhelming septicaemia should be kept in mind when any woman who is pregnant collapses unexpectedly, particularly in early pregnancy.

8. Ruptured uterus

Summary

There were three deaths from uterine rupture in this triennium as compared with four in the previous three year period. This is the lowest number recorded.

In each, some aspect of care was considered to be substandard. The continuing need to recognise the potential hazard when oxytocic drugs are used to augment labour, has again been stressed.

Ruptured uterus

In this triennium, three deaths were known to have been caused by rupture of the uterus. Unlike the previous triennium, there were no cases of rupture in which death was primarily from some other cause such as amniotic fluid embolism, postpartum haemorrhage or abortion. We have kept to the classification of uterine rupture used in the last Report and shown in Table 8.1

Trends in mortality from ruptured uterus

Table 8.1 shows that the marked decline in mortality from ruptured uterus noted in the 1979–81 Report has been sustained in the 1982–84 triennium.

Table 8.1 *Trends in mortality from ruptured uterus*

	1970–72**	1973–75	1976–78	1979–81	1982–84	Total
Scar rupture	2	1	2	0	0	5
Traumatic rupture (including rupture due to use of oxytocic drugs)	8	6	9	3	3*	29
Spontaneous rupture (without use of oxytocic drugs)	1	4	3	1	0	9
Total	11	11	14	4	3	43
Rate/million	4.8	5.7	8.0	2.1	1.6	

* One case—doubtful classification because of insufficient information.
**For changes in numbers and rates since last report see page 153.

The major cause of uterine rupture remains trauma, in particular, rupture in association with the use of oxytocic drugs. In this triennium, one case recorded as a 'traumatic' rupture occurred in a woman delivered at home by her husband. While it is possible that there was underlying trauma, there is no confirmatory evidence for this.

Traumatic rupture

The first case involved a grand multiparous woman living in poor social circumstances. She was markedly obese weighing 123 kg when delivered at term. She had an inherent dislike of doctors and had not seen her general practitioner for six years. She had a poor medical and obstetric history, having had two stillbirths and one neonatal death. In her last pregnancy, seven years before, when she delivered a stillborn baby, she was found to have gestational diabetes and hypertension with a recorded blood pressure of 240/130 mm Hg. In the present pregnancy she did not attend for antenatal care. Some weeks before delivery she developed a 'flu-like' illness for which she did not seek medical aid. One week before delivery she was reported to have collapsed and then recovered. She was delivered at home by her husband whose description suggested that the third stage was complicated by a retained placenta and haemorrhage after which she died. The baby and placenta were never seen and he said they had been 'flushed down the loo'. A Coroner's autopsy revealed a woman with no other cause for death than a laceration in a term uterus extending into the parametrium and two lacerations in the posterior vaginal fornix. There was no blood in the peritoneal cavity. The cause of the lacerations was uncertain, but the pathologist suggested they might have been caused by the hand of the patient or her husband. It seems probable that death was from haemorrhage consequent upon the lacerations.

The second case involved a young parous woman who had an uncomplicated past obstetric history of a single spontaneous vaginal delivery at term of a baby weighing 2.68 kg. She had an uneventful antenatal course except that the general practitioner suspected poor fetal growth near term. She was admitted in labour with intact membranes. After 12 hours of labour, dilatation of the cervix remained at 4 cms with the head of the fetus not being engaged and with marked caput formation. At this time the fetal heart was found to have stopped. An ultrasound scan was done to exclude placental abruption, check that the fetus was dead and exclude hydrocephaly. The haemoglobin at that time was 12 g/dl. Intravenous *'Syntocinon'* was started, and one hour later a stillborn baby again weighing 2.68kg was delivered spontaneously followed by a placenta weighing 450 g with no excessive blood loss. The mother was closely supervised by a midwife who showed her the stillborn baby. One hour later the mother vomited and was found to have blood pressure of 90/50 and a haemoglobin of 7.7 g./dl. There was no external evidence of bleeding. All efforts to resuscitate her failed and she died some hours later. A Coroner's autopsy revealed a rupture of the uterus extending from just above the cervix to the fundus with a markedly thinned lower segment, and a large amount of blood in the peritoneal cavity. There was also evidence in the left lung of amniotic fluid embolism referred to in Chapter 6. The pathologist considered that the primary cause of death was haemorrhage from the ruptured uterus.

The third case involved a multipara with an unremarkable obstetric history. Her antenatal care was uneventful and she was admitted to hospital near term with spontaneous rupture of membranes and a closed cervix. The following morning labour was induced by one tablet of prostaglandin E_2, 0.5 mg given orally. Twelve hours later she had a spontaneous delivery of a baby in good condition weighing 3.2 kg followed by a normal third stage of labour. Four hours after delivery, the patient reported feeling faint and had a tachycardia with a blood pressure of 80/50 mm Hg. She was seen by a junior doctor who ordered the end of the bed to be raised. Three hours later she was seen by the obstetrician because she "felt awful" and had pain in her right flank. He found the abdomen to be soft with minimal tenderness and no revealed blood loss, but she had a systolic blood pressure of 70 mm Hg which he ascribed to dehydration. Her blood pressure recovered to 110/70 with an infusion of 1 litre of Hartmann's solution and she was advised not to go home that day. Six hours later she collapsed on the commode with a systolic blood pressure of 90 mm Hg having passed 300–500 ml of blood clot. Blood was ordered but was not transfused because of difficulty in inserting a cannula. When she was again seen by her obstetrician, the abdominal pain was so severe and the condition of the patient so poor that the diagnosis of a ruptured uterus with a broad ligament haematoma was made and it was decided to proceed immediately with a laparotomy. The condition of the patient deteriorated rapidly, and the blood pressure could be recorded only after the rapid intravenous adminstration of fluids. At laparotomy, a large haematoma was seen on the right side of the abdomen extending from damaged uterine vessels up to the diaphragm although no rupture of the uterus could be found. Despite a subtotal hysterectomy the bleeding persisted and, even after closure of the abdomen, heavy bleeding continued through the abdominal drain. Four hours later after transfusion of 22 units of blood a further laparotomy was performed at which the right internal iliac artery was ligated. Subsequent to this a further 29 units of blood was given, but the condition of the woman deteriorated and she died 13 hours after the first laparotomy. In all, 51 units of blood were transfused and 34 units of other fluids were given, yet at no time was there evidence of a clotting defect. The coroner was not informed and an autopsy was refused by the husband.

Pathology

Two of the three cases came to autopsy. Both of these were technically satisfactory but one lacked supportive histology though this would probably not have affected the diagnosis.

Substandard care

In each of the three cases there were aspects of the care which were considered to be substandard. In the first case the pregnancy was concealed by the patient and her family until she was dead. Her failure to seek medical aid at any time in her pregnancy can be regarded only as wilful negligence on her part. In the second case the uterus was stimulated with oxytocin once the fetus was found to be dead. There is no clear evidence of when the uterus ruptured, but the presence of a dead fetus, the failure of the cervix to dilate beyond 4 cms, and a high fetal head with caput formation is evidence of obstructed labour. It would have been wise to have used oxytocin with caution rather than in a manner which led to the delivery of the fetus within an hour of starting the infusion. In the third case, earlier diagnosis and laparotomy on the basis of the presenting signs and symptoms of internal bleeding would have improved the chances of a favourable outcome. This case, as with cases described in chapters 3 and 11, yet again illustrates the importance of recognising and taking early action when signs and symptoms of concealed bleeding appear.

Discussion

All three cases of death from uterine rupture in this triennium have interesting clinical features which are educational. The case of the woman who concealed her pregnancy till death shows how important it is to remind the public of the dangers of unsupervised pregnancy and unattended home delivery. It is sometimes suggested that obstetricians over-emphasise these dangers, yet it is clear from this history that if she had sought medical care early in pregnancy her many risk factors would have been recognised and appropriate care provided.

Oxytocics were used to accelerate labour in the other two cases. The clinical history in each case was typical of 'silent' uterine rupture. In both cases the first sign was unexpected collapse from concealed haemorrhage. In one of the cases there was evidence of obstructed labour. The rapid delivery of the stillborn fetus one hour after starting intravenous *Syntocinon* suggests uterine hyperstimulation. In the other case, uterine rupture occurred sometime between the oral adminstration of one tablet of prostaglandin and delivery 12 hours later. Neither case history can be construed as an indictment of the use of oxytocics. However, Table 8.1 shows that two thirds of deaths from uterine rupture (1970–84) have been associated with the use of oxytocics, and underlines the potential hazards of these drugs. Providing these hazards are appreciated, in particular the risks to the mother and fetus of uterine hyperstimulation, oxytocics will continue to be a valuable means of treating hypotonic uterine activity. Consideration should be given to the more widespread use of electronic monitoring of uterine activity when oxytocics are being used.

9. Deaths from miscellaneous causes

Summary

This chapter describes 21 Direct deaths from miscellaneous causes in 1982–84, which cannot be easily classified. In eight instances, no cause for sudden and unexpected death could be established.

Deaths from miscellaneous causes

There were 21 Direct maternal deaths which have been coded according to the 9th Edition of the International Classification of Diseases, as follows:

ICD 181	Choriocarcinoma	1
ICD 643.1	Rupture of the oesophagus (Mallory-Weiss syndrome)	1
ICD 646.2	Post-partum haemolytic uraemic syndrome	1
ICD 646.7	Liver disorders in pregnancy	4
ICD 646.9	Sudden death of unknown cause in pregnancy	4
ICD 671.5	Cerebral venous thrombosis	3
ICD 673.0	Air embolism	1
ICD 674.8	Death due to disseminated intravascular coagulopathy (DIC) and shock lung	1
ICD 674.9	Sudden death of unknown cause in the puerperium	4
ICD 675.1	Infections of the breast and nipple associated with childbirth (purulent mastitis)	1

The cases are, however, discussed in relation to when they occurred during pregnancy, labour or the puerperium.

Rupture of the oesophagus

A woman was seen by physicians for the first time in her third pregnancy as an emergency at approximately 20 weeks gestation, with a four-week history of hyperemesis gravidarum. She had recently developed epigastric pain and haematemesis and a provisional clinical diagnosis of a tear of the oesophagus (Mallory-Weiss syndrome) was made. Whilst investigations were being carried out, the patient collapsed and had a cardiac arrest from which she did not recover. Autopsy revealed a circumferential rupture of the oesophagus, characteristic of the syndrome, with only a small continuity of tissue posteriorly.

Liver disorders in pregnancy

Cases associated with hypertensive diseases of pregnancy are counted and discussed in Chapter 2.

There were three cases of 'acute fatty liver' of pregnancy and one case of spontaneous rupture of the liver. The clinical presentation in two cases was that of disseminated intravascular coagulopathy (DIC) but autopsy revealed acute fatty liver of pregnancy. The bleeding in one instance became apparent after removal of a cervical suture, possibly causing cervical laceration; the other became obvious after Caesarean section. The diagnosis in the third case was complicated because of language difficulties. The patient was jaundiced and, delivered by Caesarean section, had a greater-than-average blood loss. This was followed by DIC and thrombocytopenia. She developed the adult respiratory distress syndrome and never recovered. Autopsy revealed an acute fatty liver.

The fourth woman collapsed with suspected placental abruption but after delivery of her stillborn infant it was obvious that there had been massive intra-abdominal bleeding. At laparotomy a spontaneous rupture of the liver was found. This was dealt with by intraoperative embolisation of the branches of the hepatic artery and packing of the liver rupture. The post-operative period was very stormy and, despite intensive care, the woman died on the 41st day after delivery.

Air embolism

This resulted during oral sexual foreplay from blowing air into the vagina of a woman who was eight months pregnant.

Sudden death of unknown cause in pregnancy

Four women died suddenly and unexpectedly at 15, 33, 35 and 38 weeks gestation, respectively. One died two hours after being seen by a midwife for a routine antenatal visit at which no abnormality had been detected. The other three women died suddenly in bed at night. One of these women did have shaking and twitching movements, but no other abnormal clinical features were reported in any of the other cases. Despite careful and detailed autopsies reviewed by other pathologists, no cause was found for any of these sudden and unexpected deaths.

Sudden deaths of unknown cause in the puerperium

Four deaths occurred at one, 14, 23 and 33 days after delivery. The first woman was admitted a few days before term with suspected, but unconfirmed, rupture of her membranes. The following day labour commenced. Progress was slow and, as she was pyrexial, delivery was by an uncomplicated Caesarean section. Five hours after delivery she was given metronidazole and ampicillin intravenously because of her continued pyrexia. Two hours later in the early hours of the morning, the patient complained of pain and was given analgesics intramuscularly. She asked for a cup of tea, then suddenly collapsed and could not be resuscitated. Despite a detailed autopsy with adequate histological material, no cause could be found for her death.

The second woman developed diplopia and hemianopia on the 14th day after an induced labour and normal delivery. She became unconscious and suffered persistent, recurrent epileptiform convulsions. Despite anticonvulsant therapy she died within 24 hours. A detailed autopsy and specialist neuropathological assessment failed to ascertain a cause for her death.

The third woman who had a long history of ulcerative colitis had not had any treatment during pregnancy. She had an emergency Caesarean section because of failure to progress in labour. The puerperium was uneventful until the 23rd day when she had a moderate attack of diarrhoea and vomiting. This became progressively worse and she collapsed and died as the ambulance arrived to take her to hospital. Although clinically suggestive of a death from ulcerative colitis, a substandard autopsy without histology revealed no specific cause of death.

The fourth woman had an uneventful first pregnancy and labour and was discharged home on her third day. Twenty nine days after delivery bronchitis was diagnosed by her General Practitioner and appropriate treatment instituted. She died suddenly on the 33rd day, without any warning symptoms. The autopsy confirmed bronchitis but revealed no cause for her death.

Other complications of the puerperium

Disseminated intravascular coagulation and shock lung

A known schizophrenic patient had late hypertension in her first pregnancy and was delivered at 38 weeks. Thirty-six hours later it was necessary to transfer her to a psychiatric unit because of her disturbed mental state. The details of events after this were limited but she had lactation suppressed with oestrogens, had two grand mal attacks, and had ECT on three occasions under general anaesthesia. She was transferred on the 18th post-partum day to a neurological centre with a diagnosis of cerebral venous thrombosis apparently confirmed by a CT scan. A chest X-ray also revealed marked patchy shadowing and it was thought that she had atypical pneumonia or shock lung. There was a progressive deterioration of her respiratory status and she was put on a ventilator the same day. She died on the 22nd post-partum day. A detailed autopsy failed to confirm a superior sagittal sinus thrombosis but revealed the typical picture of DIC and "shock lung" which has been accepted as the primary cause of death in this case.

Cerebral venous thrombosis

There were three deaths due to cerebral venous thrombosis in this triennium. Since thrombosis of cerebral veins in the absence of nasal sinus infection is extremely rare in non-pregnant women in the child-bearing age group but is well recognised in the puerperium, these deaths are counted as Direct (see also Chapter 14, page 112). One other woman who died from pulmonary embolism and is counted in chapter 4 also had sagittal sinus thrombosis.

Two deaths from cerebral venous thrombosis in the 1979–81 report were miscounted as Indirect. The only other death from this cause since 1970, counted as Direct, was in 1978. Analysis of the 6 deaths since 1978 shows that 4 of the autopsies revealed pulmonary emboli, in 5 there was evidence of thrombosis of pelvic or calf veins, and all occurred in the puerperium.

The decision as to which chapter in this report should include these deaths was made on the basis of symptoms prior to death. Since at autopsy the great majority of women who died from this cause also had thrombus present in leg or calf veins and non-fatal pulmonary emboli, consideration will need to be given in future reports as to whether these deaths, along with those from pulmonary embolism, should be included under a general heading of deaths from thromboembolism.

Post-partum haemolytic uraemic syndrome

This woman died at home 31 days after a normal delivery. She had been well until the day before her death when her husband telephoned her general practitioner's surgery because of his wife's severe headache and general malaise. The patient was dead when the doctor arrived later that day. Autopsy and histological investigations confirmed this rare diagnosis.

Puerperal breast abscess

This was an unusual case in which the patient died 14 days post-partum, after an uneventful pregnancy and delivery. She stopped breast feeding eight days after delivery. Two days prior to her death she complained of pain in her breast and since no specific abnormality was detected, the only treatment given was analgesia. She was admitted to hospital the following day with a febrile illness and 22 hours later died suddenly and unexpectedly. Autopsy with appropriate microbiological and histological analysis revealed that death had occurred as a consequence of an unsuspected staphylococcal septicaemia from a breast abscess.

Choriocarcinoma

One woman died of choriocarcinoma. She had an uneventful first pregnancy and two years later had a hydatiform mole. During follow up the levels of chorionic gonadotrophins remained high and she received four courses of chemotherapy. Twenty-two months after her hydatiform mole she became pregnant and had an uneventful pregnancy and delivery. She was discharged home on the fourth day. Eight days after delivery she was readmitted obviously very ill. Although markedly pyrexial, there were no localising signs until the 14th post-partum day when she suddenly collapsed. Laparotomy for suspected internal bleeding revealed a massive subcapsular haemorrhage of the right lobe of the liver from metastases histologically confirmed as choriocarcinoma. Secondaries were later found on chest x-ray. Despite all efforts the patient died on the 25th puerperal day. No autopsy was performed.

Pathology

All 21 deaths were counted as Direct and 20 came to autopsy. In this group, which includes unexplained deaths, the highest standards of technical, histological, microbiological and toxicological investigations are necessary and failure to perform or record them must be regarded as substandard practice. Three autopsies were regarded as technically substandard and 8 of the 20 lacked either adequate histology, microbiology or toxicology to support the diagnosis.

Comments

The deaths in this chapter fall into two groups. One consists of dangerous and rare conditions causing death, the other includes unexplained deaths. The latter group accounted for 8 out of the 21 miscellaneous deaths in this triennium compared with 6 of 14 in the last triennium. These deaths remained unexplained despite autopsies of high standard in all but one case.

The precise cause of death still cannot be identified in 3–5 per cent of all reported Direct maternal deaths and this must be accepted in the light of present knowledge.

10. Cardiac disease

Summary

There were 17 deaths from cardiac disease occurring during pregnancy or delivery or within the subsequent 42 days. This is one more than in the previous triennium. Six of these deaths (five Indirect and one Fortuitous) resulted from congenital, and 11 (nine Indirect and two Fortuitous) from acquired heart disease. Eight of the latter were due to coronary artery disease or myocardial infarction. There was one late Direct death from previous cardiomyopathy, described but not counted in this chapter.

Studies of deaths from congenital cardiac disease over the last four triennia indicate that whilst these patients are at some risk after abortions or in the last trimester of pregnancy they are particularly at risk in the puerperium. Operative or anaesthetic procedures necessary during delivery increase the dangers. These women do not adjust well to circulatory disturbances nor do they tolerate well either a relatively small blood loss or any marked degree of fluid overload. Pre-pregnancy advice and expert care throughout pregnancy are essential for them and their special cardiological problems must be appreciated by those who look after them at delivery and in the puerperium.

The risk of death from acquired heart disease, which increases with age, particularly after the age of 30 years; is now predominantly due to coronary arterial narrowing or thrombosis rather than rheumatic origins. Death was rarely preceded by premonitory symptoms such as angina.

Cardiac disease

In this triennium there were 17 deaths from cardiac disease during pregnancy or within 42 days after delivery. This compares with 16 and 17 respectively in the two previous triennia.

In the 1979–1981 report, deaths from cardiomyopathy arising in pregnancy were regarded for the first time as Direct deaths. The only death which occurred from this cause in the present triennium was a late death, from heart failure, eight weeks after delivery, of a woman who had had cardiomyopathy diagnosed during a pregnancy four years earlier. Despite this, she had a second child one year later and there were no complications either then or in her third and final pregnancy and delivery. All cardiac deaths which occurred in this triennium are therefore coded as Indirect or Fortuitous. The precise coding of deaths certified as due to coronary insufficiency remains difficult. It can be argued that all should be regarded as Fortuitous, but since there are physilogical circulatory changes during pregnancy and after delivery which might adversely affect the cardiovascular system, we hold the opinion that each death should be considered individually. An analysis of the 17 cardiac deaths is given in Table 10.1, and of the deaths from congenital and acquired disease over the last four triennia in Table 10.2.

Table 10.1 *Analysis of cardiac deaths, 1982–84*

	Direct	Indirect	Fortuitous	Total
Congenital	–	5	1	6
Acquired				
Coronary disease or Myocardial infarction	–	6	2	8
Cardiomyopathy	–	–	–	–
Others	–	3	–	3
Total	–	14	3	17

The number of deaths from congenital and acquired heart disease from 1973–1984 are shown in Table 10.2.

Table 10.2 *Number of maternal deaths from congenital and acquired disease during 1973–84*

	Acquired	Congenital	Total
1973–75	14	4	18
1976–78	14	3	17
1979–81	12	4	16
1982–84	11	6	17
Total	51	17	68

Congenital heart disease

Six deaths were associated with congenital heart disease; five were considered Indirect and coded to ICD 648.5, and one Fortuitous and coded to ICD 746.8.

Eisenmenger's syndrome

Four of the five Indirect deaths were associated with Eisenmenger's syndrome. This syndrome occurs in patients who have congenital atrial or ventricular septal defects or possess a persistent patent ductus arteriosus. Most have had a left-to-right shunt since birth. This can be asymptomatic, but can lead in adolescence to increasing pulmonary hypertension with eventual reversal of blood flow and the development of a right-to-left shunt. Clinically they have cyanosis, pulmonary hypertension, right ventricular hypertrophy and a liability to sudden death, especially in the puerperium. All five patients had been under expert cardiological care at some time and three had had some form of surgical correction with varying degrees of success. All appeared to have received medical advice about the risks associated with pregnancy and all were pregnant for the first time.

One woman who had an inoperable defect and intermittent cyanosis had a relatively uneventful pregnancy and vaginal delivery. On the following day a bleeding vaginal tear was repaired under general anaesthesia. Two days later she collapsed suddenly and could not be resuscitated. Blood loss was a major factor in precipitating her cardiac failure. Another woman continued with her pregnancy despite medical advice. She became increasingly dyspnoeic and cyanosed in the third trimester and developed signs of cerebral anoxia. She had a Caesarean section at 31 weeks gestation because of her deteriorating condition and died seven hours after delivery. No autopsy was performed. A third woman died from massive retroperitoneal and intrapelvic haemorrhage following abdominal tubal ligation associated with a legal abortion and is mentioned in chapter 11. A fourth woman, who had had a repair of a ventricular septal defect as a child, which had subsequently partly broken down, had been advised against pregnancy. After excellent medical and obstetric care she went into spontaneous labour at 37 weeks and had an elective outlet forceps delivery. She initially developed undetected intrauterine bleeding and despite adequate fluid replacement died from irreversible cardiac failure. The fifth woman, who had had a partially successful closure of a ventricular septal defect and a patent foramen ovale, should probably also be considered as having Eisenmenger's syndrome. She had had one previous termination at 15 weeks and had been strongly advised against further pregnancy. She first presented at 24 weeks in her second pregnancy and was adjudged too far advanced for termination. She died at home some two weeks later; no autopsy was performed but death was certified as due to heart failure.

It is important to appreciate that comparatively small changes in circulatory volume, resulting either from haemorrhage or fluid overloading, which might be insignificant in normal women, can be critical in women with Eisenmenger's syndrome.

Congenital complete heart block

The Fortuitous death of this woman was unusual in that she had a mesothelioma of the atrioventricular node thought to have been present since birth. She died 26 days after a termination at 13 weeks and is mentioned in Chapter 11.

Acquired heart disease

Eleven deaths were associated with acquired heart disease; nine were considered as Indirect deaths and were coded to ICD 648.6 and two as Fortuitous and coded to ICD 410. The distribution by age and parity of maternal deaths over the last four triennia is shown in Table 10.3.

Five of the eight Indirect deaths were attributed to coronary arterial disease or to myocardial insufficiency. The death rate from coronary artery disease per million maternities for the triennia 1973–75, 1976–8, 1979–81 and 1982–4 has been 2·1, 3·4, 3·1 and 2·7 respectively. Only two women had premonitory

Table 10.3 *Number of maternal deaths from acquired cardiac disease, by age and parity*, 1973–84*

Age (years)	1973–75	1976–78	1979–81	1982–84	Total
Under 25	3	5	4	0	12
25–29	5	3	1	2	11
30–34	1	1	2	4	8
35 and over	5	5	5	5	20
Total	14	14	12	11	51
Parity					
1	2	6	2	1	11
2	5	2	5	3	15
3	4	3	2	2	11
4 and over	3	3	3	4	13
Not stated				1	1
Total	14	14	12	11**	51**

*For definition of parity see page 154.
**Includes 1 not stated

symptoms. The first woman aged 38 years, who complained of anginal pain when 35 weeks pregnant, was found to have a normal ECG and discharged home where she collapsed and died undelivered, three days later. Ischaemic heart disease due to coronary atheroma was confirmed at autopsy. The second woman was admitted as an emergency in severe shock and was clinically aborting. She also had had chest pain which after her death was reported to have been actually present for 24 hours. Autopsy revealed that she had aborted a cervical pregnancy but of more significance was severe coronary artery atheroma and occlusion of the right coronary artery. Because of the obvious abortion ergometrine had been given and this might well have had an adverse effect on the coronary circulation. A third woman who died following a coronary thrombosis had severe diabetes mellitus. Another, who had suffered for many years from severe juvenile diabetes with renal, cerebral and terminal cardiac lesions died 35 days after delivery. The main cause of death was considered to be diabetes and she is counted in Chapter 14.

Four of the Indirect deaths were associated with other forms of acquired heart disease. A woman aged 34 years died suddenly, 20 days after delivery by emergency Caesarean section. She was thought clinically and at autopsy to have had a thrombus in the anterior descending branch of the left coronary artery but histology showed the lesion to be a small dissecting aneurysm with associated cystic medial arterial degeneration. This type of aneurysm is exceedingly rare in coronary arteries, but examples both in coronary and splenic arteries in pregnancy are well recorded. A second woman aged 38 years died five days after forceps delivery of her third baby; death was due to myocarditis. A third woman, 34 years of age and 32 weeks into her 6th pregnancy, had a history of rheumatic cardiac disease. She did not present for antenatal care until 19 weeks pregnant and was always a poor attender who constantly changed her address. She was brought into hospital dead and autopsy revealed a severe degree of mitral stenosis and aortic incompetence.

The cause of death in the final case, a woman, aged 27 years, who had had rheumatic fever as a child was difficult to assess. She went into heart failure with atrial paroxysmal tachycardia in the 21st week of her first pregnancy. Thereafter she remained in hospital and had a low forceps delivery at 39 weeks gestation. On the 7th post-partum day her tachycardia recurred and she became hypotensive; she subsequently developed disseminated intravascular coagulation, thrombocytopenia, haemolytic jaundice and anuria, went into coma and died on the 30th post-partum day. The suspected cause of death was sepsis, but the autopsy was of poor quality and no review was possible.

Two deaths were considered to be Fortuitous; in each the cause of death was myocardial infarction.

'Time of death'

The time of death from heart disease associated with pregnancy in relation to each trimester and to delivery or abortion is given for the years 1973–1984 in Tables 10.4 and 10.5.

Table 10.4 *Gestational age of women at time of death from congenital heart disease 1973–84 and interval between abortion or delivery and death*

Weeks pregnant	Died before delivery	Died after abortion or delivery	Total deaths	Interval between delivery or abortion and death		
				Under 24 hours	1–7 days	8–42 days
8–19	1	2	3	–	–	2
20–27	1	–	1	–	–	–
28–35	–	2	2	–	1	1
36–42+	–	11	11	1	5	5
Total	2	15	17	1	6	8

Table 10.5 *Gestational age of women at time of death from acquired heart disease 1973–84 and interval between abortion or delivery and death*

Weeks pregnant	Died before delivery	Died after abortion or delivery	Total deaths	Interval between delivery or abortion and death		
				Under 24 hours	1–7 days	8–42 days
8–19	6	3	9	3	–	–
20–27	7	1	8	–	1	–
28–35	2	6	8	–	3	3
36–42+	3	23	26	2	6	15
Total	18	33	51	5	10	18

Women with congenital heart disease are at greatest risk in the puerperium or after abortion (15 out of 17 deaths) rather than during pregnancy (2 deaths out of 17).

Women with acquired heart disease are at risk throughout pregnancy (18 deaths out of 51) and the puerperium (33 deaths out of 51) but not especially in the last trimester (5 deaths out of 18).

Pathology

There were 17 deaths from cardiac disease of which 14 were Indirect; three were Fortuitous and are not further described here. Of the 14, five resulted from congenital heart disease and three of these came to autopsy. All were anatomically unsatisfactory in that the congenital anomalies present were not fully described and histology was not available. The remaining nine resulted from acquired heart disease and eight came to autopsy. One autopsy was considered anatomically substandard because description of the coronary arteries was inadequate.

Substandard care

Two patients failed to attend for antenatal care and one was not followed up by administrative staff. There appears to be a generalised lack of awareness that the risk for cardiac patients is greatest in the puerperium and that patients with congenital cardiac lesions do not tolerate small blood losses or circulatory overload which might be less serious in healthy women.

Conclusions

More women with congenital heart disease or heart disease acquired in childhood are now surviving to childbearing age and it is important that they receive proper and timely pre-pregnancy advice and assessment. The surgical correction of a congenital lesion is often of necessity incomplete and any such lesion, corrected or not, leaves the patient at risk. It has to be accepted, however, that there will be a small number of patients who will not accept professional advice. Despite specialised antenatal management, in which a cardiologist should also be concerned, abortion or delivery and the early puerperium pose serious problems particularly if operative procedures and anaesthesia are necessary. Only two of the women who died from congenital heart disease after delivery had uncomplicated deliveries. It is equally essential to provide the same sort of clear and adequate advice for the future for those whose pregnancy has a successful outcome. Special care is needed during the puerperium; small blood losses are not well tolerated and equally it is easy to produce right heart failure with minor degrees of circulatory overload following transfusion.

The majority of patients who die from acquired heart lesions due to coronary artery disease do not have premonitory symptoms and there appears to be no means, short of routine cardiographic examination of all pregnant women over the age of 35 years, of forecasting this complication.

11. Abortion

Summary

There were 11 Direct obstetric deaths from abortion in 1982–84, detailed in Table 11.2. This was three fewer than in the last triennium. Four deaths were associated with spontaneous abortion (ICD 634) and seven deaths with legal abortion (ICD 635). For the first time no deaths were attributed to illegal abortion. Two other obstetric deaths were considered to be due to complications of anaesthesia. One followed a legal abortion and another occurred during preparations for evacuation of retained products of conception. These have been counted as direct anaesthetic deaths and are discussed in Chapter 13.

Following the precedent of previous triennia, two women with hypertensive diseases of pregnancy who had their pregnancy terminated before 28 weeks of gestation because of eclampsia (ICD 642.6), have been excluded from this chapter and included in Chapter 2. These women could be regarded as having had abortions, (as neither fetus was born alive) but have been counted only in Chapter 2 as Direct obstetric deaths. This same classification was made in 1979–81 for 5 cases, 2 of pre-eclampsia and 3 eclampsia.

There were 4 Indirect deaths, 3 related to legal and one to spontaneous abortion. There were also 5 other deaths, 3 following legal abortion and 2 following spontaneous abortion which were considered to be unrelated to the pregnancy and classified as Fortuitous. These are listed with other Fortuitous deaths in Chapter 15.

Introduction

Table 11.1 shows that the rate of death from abortion in 1982–84 was 5·8 per million maternities and 4·4 per million pregnancies, which are the lowest recorded so far. The major change in mortality from legal abortion occurred between 1970–75 when there was a fall from 28 to 9 deaths. Since then the number has remained fairly constant. The most dramatic change over these years has been the consistent decline in deaths from illegal abortion up to the present. In 1970–72, when the steep increase in the number of legal abortions being done had levelled out, there were 37 deaths from illegal abortion. Nine years later, in 1979–81 there was one, and in this triennium there was none. Deaths from spontaneous abortion fell sharply between 1967–69 when there were 25, to 1970–72 when there were 6, since when the number has remained fairly constant.

Table 11.1 *Direct abortion deaths in the triennial reports 1970–84 by type of abortion with estimated rates per million maternities and estimated rates per million pregnancies*

Type of abortion		*1970–72*[6]	1973–75[6]	1976–78	1979–81	1982–84	1970–84
Spontaneous		6	4	2	6	4	22
	Spontaneous	4	3	2[3]	4[7]	4[5]	17
	Missed	2	1	–	2	–	5
Legal		30[1]	13[2]	8[3]	5[4]	7[5]	63
Illegal		37	10	4	1	–	52
Unspecified		–	–	–	2	–	2
Total		73[1]	27[2]	14[3]	14[4]	11[5]	139
Rates per million maternities		31·8[1]	14·1[2]	8·0[3]	7·3[4]	5·8[5]	14·2
Rates per million pregnancies		25·3[1]	10·5[2]	6·0[3]	5·5[4]	4·4[5]	10·8

[1] Does not include 7 anaesthetic deaths associated with legal abortion.
[2] Does not include 1 anaesthetic death associated with legal abortion.
[3] Does not include 5 anaesthetic deaths, 4 associated with legal and 1 with spontaneous abortion.
[4] Does not include 1 anaesthetic death associated with legal abortion.
[5] Does not include 2 anaesthetic deaths, 1 associated with legal and 1 with spontaneous abortion.
[6] For changes in numbers and rates since last report see page 153.
[7] One other death is known to have followed a spontaneous abortion in the 1979–81 triennium, but it was reported too late to be included in the Enquiry.

Table 11.2 shows details of individual cases of Direct deaths. The commonest single cause of death amongst the legal abortion cases (7) was haemorrhage which in all three women was initially unrecognised and are also mentioned in Chapter 3.

Details of individual Indirect cases are given in Table 11.3. Three of the cases are described more fully and counted in Chapter 14 and one in Chapter 10.

Table 11.4 summarises the number of women who died from all kinds of abortion between 1970–84 relative to age. The figures confirm the known association between age and death from spontaneous and legal abortion, women of more than 35 years being at much greater risk.

Table 11.5 summarises the number of women who died from all kinds of abortion between 1970–84 relative to parity. The risk of death is distributed evenly throughout the parities although this does not take into account the distribution of parities amongst all maternities. From this it can be concluded that the increased risk of death amongst women of more than 35 years is unlikely to be affected by parity.

Table 11.2 *Direct deaths associated with abortion, 1982–84*

Number	Duration of pregnancy (weeks)	Age (years)	Parity	Method of abortion or operation if performed and complications	Cause of death	ICD Code	Other chapter(s)
Legal abortion							
1.	7	<35	1	Dilatation and curettage	Pulmonary embolism	635.6	4
2.	12	<35	3	Vacuum aspiration. *Perforation of uterus, haemorrhage*	Haemorrhagic shock	635.1	3
3.	12	>35	4	Vacuum aspiration & laparoscopic sterilisation. *Perforation of uterus, haemorrhage*	Haemorrhagic shock	635.1	3
4.	14	>35	4	Extra-amniotic prostaglandin	Pulmonary embolism	635.6	4
5.	17	<35	2	Dilatation and curettage. *Perforation of uterus, laparotomy, haemorrhage*	Haemorrhagic shock	635.1	3
6.	17	>35	5	Intra-amniotic prostaglandin	Pulmonary oedema	635.7	
7.	25	<35	1	Hysterotomy	Pulmonary embolism	635.6	2 and 4
Spontaneous abortion							
8.	12	<35	3	Evacuation of retained products. *Laparotomy, DIC and sepsis*	Spontaneous abortion with sepsis	634.0	7
9.	14	<35	2	*Sepsis*	Spontaneous abortion with sepsis	634.0	7
10.	22	<35	3	Evacuation of retained products	Spontaneous abortion with sepsis?	634.0	7
11.	27	<35	1	*Cardio-respiratory collapse, cardiac arrest*	Bronchopneumonia (terminal)	634.7	

Table 11.3 *Indirect deaths associated with abortion, 1982–84*

Number	Duration of pregnancy (weeks)	Age (years)	Parity	Method of abortion or operation if performed and complications	Cause of death	ICD Code	Other chapter(s)
Legal abortion							
1.	10	<35	1	Vacuum aspiration and sterilisation by tubal ligation. *Haemorrhage*	Eisenmenger's syndrome	648.5	10
2.	11	>35	5	Vacuum aspiration and laparoscopic sterilisation	Gas embolus (at laparoscopy)	(648.9+998.8)	14
3.	19	<35	2	Hysterotomy	Fat embolus, systemic lupus erythematosus	(648.9+710.0)	14
Spontaneous abortion							
1.	25	<35	2	Spontaneous abortion	Haemorrhagic cerebral infarction	674.0	14

Table 11.4 *Number of women in the enquiry who died from abortion and estimated death rates per million pregnancies, 1970–84**

Age (years)	Spontaneous and other abortions	Legal abortions	Illegal abortions	All abortions	Estimated rate per million pregnancies
Under 25	10	19	21	50	8·2
25–34	6	14	23	43	7·4
35 and over	8	30	8	46	49·3
All ages	24	63	52	139	10·8

*For changes in numbers and rates since last report see page 153.

Table 11.5 *Number of women in the enquiry who died from abortion by parity† 1970–84‡. Figures in parentheses are distribution of parity for all maternities during this period.*

Parity	Spontaneous and other abortions	Legal abortions	Illegal abortions	All abortions	% of all abortions
1	5	14	13	32	23 (40)*
2	4	10	9	23	17 (35)
3	8	10	14	32	23 (15)
4	3	11	7	21	15 (6)
5 and over	3	17	6	26	19 (4)
Not stated	1	1	3	5	4
Total	24	63	52	139	100 (100)

* Figures in parentheses relate to true birth order.
†For definition of parity see page 154.
‡For changes in numbers and rates since last report see page 153.
Note—a maternity denotes a pregnancy which resulted in the birth of one or more live or still born children, ie, it excludes abortions.

Table 11.6 summarises the number of women who died from all kinds of abortion between 1970–84 relative to marital status. As expected, the number of abortions is somewhat higher amongst the unmarried group of women compared with the married. However, the number of spontaneous abortions is surprisingly high amongst married women and gives rise to the speculation that some of these could have been illegal abortions which were classified as spontaneous abortions because of lack of evidence.

Table 11.6 *Marital status of women who died from legal and spontaneous abortion in 1970–84*. Figures in parentheses refer to distribution of married and unmarried women aged 16–44 years for all maternities during this period.*

Status	Spontaneous abortions	Unspecified abortions	Legal abortions	Illegal abortions	Total	% of abortions
Married	20	2	42	31	95	68 (89)
Single, divorced or widowed	2	–	21	21	44	32 (11)
Total	22	2	63	52	139	100 (100)

*For changes in numbers and rates since last report see page 153.

Legal abortion

Factors predisposing to death from legal abortion—Haemorrhage

Three of the seven Direct deaths were due to abdominal haemorrhage. A further woman with congenital Eisenmenger's defect of the heart, counted as an Indirect death in Chapter 10, had abdominal haemorrhage as a complication of concurrent sterilisation. In three cases, the bleeding was sudden and severe with death occurring within a few hours. In the fourth, bleeding was more chronic and death occurred after several days. There are important lessons to be learnt from these cases.

Failure to recognise the gravity of accumulated blood loss from a perforation of the uterus was a major factor leading to death. In the first case, the blood loss had been so great that the systolic blood pressure was as low as 70 mm Hg (within 25 minutes of starting the vacuum aspiration). The gynaecologist decided to go ahead with an exploratory laparotomy having infused Hartmann's solution and then plasma to try to correct the circulatory collapse. Blood had been ordered but arrived more than an hour after the start of the operation. By that time, the patient had lost a further estimated 1500 mls of blood. Six units of unwarmed blood were eventually given but the condition of the patient continued to deteriorate until she had a cardiac arrest.

In the second case, a vaginal termination of a mid-trimester pregnancy was performed under general anaesthesia. The gynaecologist recognised that the uterus had been perforated, but decided to observe the patient. He left the hospital without ordering blood to be cross matched or informing the duty consultant gynaecologist. There were no medical staff in the hospital. Three hours later the patient collapsed and, although a drip was started, still no blood was ordered. Two hours later the gynaecologist returned and decided to proceed to laparotomy. A blood transfusion was commenced one hour after the start of the operation during which laceration in the uterus was repaired. By this time the patient's shock was irreversible and she died 90 minutes later.

In the third case, brisk vaginal bleeding occurred after vacuum aspiration of a first trimester pregnancy. The consultant gynaecologist, convinced that the bleeding was due to failure of the uterus to contract, controlled the bleeding by uterine massage. He then proceeded to laparoscopic sterilisation at which time a comment was made that large vessels were seen in the broad ligament. In retrospect, it seems likely that this was a developing haematoma but, in the event, no intraperitoneal bleeding was reported. The post-operative supervision of the patient was by a junior doctor who noted tachycardia and hypotension but took no further action. That evening the patient collapsed. At laparotomy, when the patient was moribund, intraperitoneal bleeding was found due to a high laceration of the cervix.

These three cases illustrate the danger of failing to maintain the intravascular blood volume by adequate replacement in the presence of continuous bleeding. In two of these women, intravenous fluids were being given but it seems likely that they were still hypovolaemic. It was clearly hazardous to embark on a laparotomy under general anaesthesia without adequate transfusion. The decision as to whether or not to proceed with a major surgical procedure in the presence of heavy and prolonged blood loss is dependent on the general condition of the patient. The assessment of this must be the joint responsibility of gynaecologist and anaesthetist.

In the other case, it is likely that death could have been avoided by appropriate resuscitation and haemostasis, if the signs of concealed bleeding had been recognised earlier.

Lack of availability of facilities

In three Direct deaths due to haemorrhage there was evidence that the facilities and/or procedures for dealing with emergencies were substandard. The lack of 0 negative blood on site was crucial. One of the deaths was in an NHS hospital and the other two were in nursing homes approved by the Secretary of State under the Abortion Act (1967). Effective procedures for obtaining blood in an emergency have always been one of the requirements for approval. However, in 1985, the Department issued further 'Guidelines on Emergency Blood Cover' to all approved nursing homes (see Appendix). Since then, strict adherence to the Guidelines, which were revised in 1986, has been made a condition of approval or re-approval for all nursing homes admitting patients for abortion, and these are checked by regular inspections. Clearly, such a code of practice should apply to all NHS institutions.

Operative technique

One death followed the intra-amniotic injection of prostaglandin. The recommended technique in the unit for the intra-amniotic injection of PGF_{2a} was to withdraw clear liquor before slow injection of the drug. This was adhered to, but between five and ten minutes after the injection the patient complained of "feeling funny" and went into cardiac arrest from which she eventually died. It seems likely that the prostaglandin, which was more concentrated than that normally used intravenously, was inadvertently injected into a maternal venous sinus. Despite the fact that the technique prescribed was being adhered to, injecting any potentially toxic substance into the amniotic sac through a needle over more than a few seconds must carry an increased risk of intravenous injection. Ideally, such an injection should be made through a fine polythene tube which has been fed through a needle into the amniotic cavity.

The second death followed the legal abortion of an unwanted pregnancy which was the result of an ineffective sterilisation. Death occurred with the extension of a massive thrombosis of calf veins leading to bilateral occlusion of the pulmonary arteries. This case is described in Chapter 4 on pulmonary embolism.

Concurrent sterilisation

Two of the three deaths following sterilisation performed immediately after the vaginal termination of pregnancy are counted as Indirect deaths. One of these occurred in a woman in the first trimester after an uneventful vacuum aspiration under general anaesthesia. At laparoscopy, on introducing the Verre's needle suprapubically, gas was noted to be coming out through the cervix. The needle was immediately withdrawn and reinserted and pneumoperitoneum achieved. However, as the laparoscope was introduced, cardiac arrest occurred. Following resuscitation, the woman was transferred to the intensive care unit where she died seven days later. The second death, which is counted in Chapter 10, was a case of intraperitoneal bleeding after tubal ligation in a woman who was being treated with anticoagulants. The third death occurred in a parous woman after an uneventful first trimester abortion by curettage. The gynaecologist then proceeded to a mini-laparotomy in order to sterilise the patient. The operation was prolonged because of obesity leading to difficulty in entering the abdomen. As the abdomen was being closed, cardiac arrest occurred due to an anaesthetic complication and the case is counted as a Direct death in Chapter 13.

Sterilisation at the time of legal abortion is still a relatively common procedure. In 1982–84 it was carried out on 18,461 resident women in NHS institutions and 4590 such women in non-NHS institutions in England and Wales. The procedure is more dangerous than in the non-pregnant woman for a number of reasons. The uterus is larger and thus more liable to be damaged by the Verre's needle or a trochar during laparoscopy. Bleeding or gas embolism are more likely to occur because the pelvis and its structures are much more vascular. Finally, the combined operation, regardless of whether laparoscopy or laparotomy is used for sterilisation, takes longer than a straightforward vacuum aspiration and what starts as a simple procedure becomes more complicated. The merit of carrying out sterilisation at the time of legal abortion is that the patient has only one anaesthetic. Against this benefit are the risks of the combined operation and the acknowledged fact that a proportion of women who request sterilisation before legal abortion change their minds.

Ideally, sterilisation should be deferred for several weeks after legal abortion (interval sterilisation). This is not always possible for good reasons, so it would not be justified to make a strong recommendation that concurrent sterilisation should be proscribed. We would recommend that the procedure should always be performed by an experienced gynaecologist and an anaesthetist because of the potential hazard of sterilisation concurrent with legal abortion, regardless of the techniques employed.

Substandard care

Consideration of the case histories suggests that care was substandard in 4 of the 7 Direct deaths, and in 3 of the 4 Indirect deaths.

Spontaneous abortion

There were four deaths directly attributed to a spontaneous abortion in this triennium. In the three cases described in Chapter 7 there was severe sepsis with no evidence of criminal interference.

The fourth death was that of a woman with threatened premature labour, treated at 26 weeks gestation with betasympathomimetic drugs and steroids. After a week it was clear that she had an intrauterine infection and that labour could no longer be suppressed, so all treatment was stopped and labour allowed to progress. She collapsed a few hours later and after resuscitation had to be maintained on artificial ventilation. Twenty-three days later she died. Although the terminal cause of death was pneumonia, the cause of the original collapse was not clear. After autopsy, the most likely course of events was thought to be cardio-respiratory arrest with resultant hypoxia and irreversible brain damage. Betasympathomimetic drugs and septicaemia could not be excluded as contributory factors as there was no evidence of amniotic fluid embolus. The association of severe cardiac dysfunction leading to collapse with the use of these drugs in the treatment of threatened preterm labour, is noteworthy. A similar comment was made in the report of the last triennium (p123).

Pathology

Of the 11 deaths counted as Direct, nine came to autopsy. No autopsy was considered anatomically substandard, but it is remarkable that one case in which an autopsy was not performed was not reported to the coroner. No record of histology was available in three but in only one of these was it necessary to confirm the anatomical diagnosis. Microbiological examinations made either during life or at autopsy were satisfactory.

Fortuitous deaths (Table 11.7)

There were five deaths from a condition not directly linked with the pregnancy. These are also listed in Chapter 15.

Table 11.7 *Fortuitous deaths following abortion*

Legal abortion There were three Fortuitous deaths following legal abortions:—	
1. Bronchopneumonia in patient with multiple sclerosis	(ICD 485x)
2. Mesothelioma of atrioventricular node (congenital heart disease)	(ICD 746.8)
3. Manslaughter	(ICD (80 4x) (E968.2)
Spontaneous abortion There were two Fortuitous deaths following spontaneous abortion:—	
1. Aplastic anaemia	(ICD 284.9)
2. Cerebral infarction	(ICD 434.9)

Addendum

This case was reported too late to be included in the main enquiry and the Tables but is mentioned because of its interest.

The patient was a multigravida who had two full-term deliveries both with insertion of a Shirodkar suture, and one preceding stillbirth at 28 weeks gestation. In the index pregnancy, a cervical suture (MacDonald) was inserted at 14 weeks. Moderate vaginal bleeding occurred and eventually, an offensive vaginal discharge, which was treated with Neomycin pessaries. At 17 weeks a further cervical suture was inserted uneventfully, the first one having cut out. At 22 weeks she was admitted with ruptured membranes and the following day had a pyrexia of 40°C and a rigor. She was treated with antibiotics and the suture removed by a registrar under general anaesthesia. Uterine contractions were stimulated with *Syntocinon*. Unfortunately, the placenta remained adherent, so an attempt was made by the registrar to remove it manually in the ward. During this procedure, catastrophic bleeding commenced, so the patient was moved to the theatre where cardiac arrest occurred. The patient was resuscitated but bleeding persisted, so a hysterectomy was done by the consultant. The uterus was found to be perforated. The patient was moved to an intensive care unit in a nearby hospital but never regained consciousness. At autopsy, a placenta accreta was found in the lower part of the uterus associated with extensive inflammation of the myometrium. Death resulted from catastrophic bleeding from placenta praevia which had been suspected from an ultrasound scan. The fact that it was a placenta accreta undoubtedly increased the risk of uncontrollable haemorrhage. However, the attempts to remove the placenta from the uterus in the ward must be regarded as substandard care, particularly in the presence of such severe uterine infection.

Conclusions

1. The few deaths relative to the large numbers of legal abortions carried out in both the NHS and private sectors are evidence that surgical techniques in current use are satisfactory. Nevertheless, the fact that in this triennium there was evidence of substandard care in seven cases, suggests that constant vigilance must be exercised to maintain the standards of clinical care of this potentially dangerous procedure. Particular points which have been noted in this triennium are:—

(a) blood for transfusion must be readily available in NHS and private hospitals where legal abortions are carried out.

(b) the clinical skills of gynaecologists and anaesthetists undertaking legal abortions or concurrent sterilisation in the NHS and private sectors must be of the highest order, particularly when (as in one case) a vaginal termination of a second trimester pregnancy is attempted.

(c) suspected damage to the genital tract demands careful observation for at least 24 hours after the operation, and earlier rather than later surgical intervention when occult bleeding is suspected.

(d) the practice of sterilisation concurrent with abortion should be done only for good reason.

2. There were no deaths in this triennium attributable to illegal abortion, which could be regarded as the hoped for effect of the 1967 Abortion Act.

3. Septicaemia leading to collapse, though rare, continues to be a threat to all women at any stage of pregnancy but particularly at the time of abortion.

Annexe to Chapter 11

Guidelines for emergency blood cover at nursing homes approved for abortion by the Secretary of State

One of the requirements for places to be approved under the Abortion Act 1967 is that proper arrangements have been made to deal with any emergencies arising in the treatment of patients for termination of pregnancy. A very important part of these arrangements is the emergency provision of blood, blood products and other replacement fluids and the availability of the necessary blood tests before operations. The following guidelines represent the *minimum* standard of emergency blood cover acceptable to the Department.

Blood supplies and other IV fluids required in an emergency

1. *Available immediately at the Nursing Home*

Plasma Protein Fraction (Minimum 2 units of 500 ml)
Degraded Gelatin Solution (4 litres)
Hartmann's Solution
Dextrose saline

2. *Available immediately or within 15 minutes of requirement*

a. *For all cases* two units of O Rhesus-negative blood available for use *within 15 minutes* (either held at the Nursing Home or "ear-marked" for the Nursing Home and held in an adjacent hospital blood bank or private laboratory).
b. *Either:*
In addition for *special risk cases* (for example those of 16 weeks gestation and above). Two units of cross-matched blood held at the Nursing Home, Hospital blood bank or private laboratory and available within 15 minutes;
Or:
i. *For cases from 16–20 weeks* gestation provided a sample of blood has been taken in advance from the patient and sent to the servicing laboratory and has been grouped and screened for red cell antibodies, the abortion can proceed without cross-matched blood being available *provided that* clinically significant antibodies have not been detected. In this situation any blood required can be the Group O Rhesus-negative blood held at the Nursing Home and further cross-matched blood can be supplied (under Section 2 of the Guidelines) within 60 minutes. This could be expedited by a rapid cross-match in the knowledge that antibodies were absent. If antibodies *were present*, then 2 units of blood for this case should be cross-matched in advance *before the abortion* is performed.

ii. *For cases over 20 weeks* If suitable blood for the patient could not be guaranteed within 15 minutes, then 2 units should be cross-matched prior to the abortion.

3. *Available if the emergency continues*
a. Supplies of cross-matched blood should be 'rapidly obtainable' in an emergency (not more than 60 minutes). This time should take into account geographical distance and travelling conditions at the busiest times.
b. If the Nursing Home is more than 15 minutes travelling distance away, (in one direction) serum should be held in advance at the hospital blood bank or private laboratory for all cases, for cross-matching if required.

3. If the emergency blood supplies available to the Home have been used up the operation list should be suspended until they have been replaced.

4. The Nursing Homes should have a suitable blood refrigerator solely reserved for blood storage, and any supplies of blood should be supervised by a haematologist for recycling if possible.

5. Blood grouping of the patients before operation should usually be performed by the hospital or private laboratory which can provide a 24 hour service for cross-matching and hold serum samples if required.

Department of Health and Social Security
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12. Caesarean section

Summary

There were 69 deaths amongst women delivered by Caesarean section during 1982–84 as compared with 87 in 1979–81. There were 44 Direct deaths, 20 were Indirect and 5 Fortuitous. The fatality rate for all deaths following Caesarean section in NHS hospitals in England and Wales continues to decline being 0·37 per 1000 Caesarean sections as compared with 0·52 (1979–81), 0·66 (1976–78), 0·76 (1973–75) and 1·01 (1970–72).

Despite the national increase in the number of Caesarean sections done in this triennium, Direct deaths have again fallen.

The indications for which the Caesarean sections were done and the immediate causes of death show little change from the previous triennium. The major indications were hypertensive diseases of pregnancy in its various forms (33%), followed by delay in labour (19%), fetal distress (14%) and antepartum haemorrhage (14%).

The ratio of Direct deaths attributable to emergency and elective Caesarean sections is 4·5:1 which is less than that for the last triennium.

Care was thought to be substandard for 23 of the 44 Direct deaths (52%).

Caesarean section

Table 12.1 shows the distribution of deaths classified according to the type of death for each triennium from 1970. The total of 69 deaths in this triennium is made up of 44 Direct maternal deaths, 20 Indirect and 5 Fortuitous deaths.

Table 12.1 *Deaths connected with Caesarean section, 1970–84*

	Total maternal deaths	Direct deaths	Indirect deaths	Fortuitous deaths	'Associated'* deaths
1970–72	102	81	–	–	21
1973–75	77	60	–	–	17
1976–78	80	61	14	5	–
1979–81	87	59	25	3	–
1982–84	69	44	20	5	–

*Associated deaths subdivided into Indirect and Fortuitous since 1976.

In order to determine the risk of death amongst women delivered by Caesarean section it is necessary to know the number of operations performed in the triennium. An estimate of the number of operations performed in NHS institutions in England and Wales during 1982–84 has been obtained from the Hospital In-Patient Enquiry (HIPE). These figures and the calculated fatality rates for the triennia since 1970 are shown in Table 12.2:

Table 12.2 *Estimated number of Caesarean sections performed, and estimated fatality rate† per thousand Caesarean sections within 42 days in NHS hospitals in England and Wales for each triennium, 1970–84*

	1970–72	1973–75	1976–78	1979–81	1982–84	Total
Total maternities in NHS hospitals	2,000,612	1,799,980	1,689,670*	1,876,570*	1,840,970	9,270,802
Estimated number of Caesarean sections	103,310	101,410	120,570	167,020	185,820	678,130
Percentage of maternities by Caesarean sections in NHS hospitals	5·2	5·6	7·1*	8·9*	10·1	7·3
Deaths after Caesarean sections (Direct maternal and "Associated" deaths from enquiry series)	102	77	80	87	69	415
Estimated fatality rate per thousand Caesarean sections	0·99	0·76	0·66	0·52	0·37	0·61

*Revised figures to those previously published; corrected for error in calculation of total maternities in NHS hospitals.

†Fatality rate for all deaths after Caesarean section, Direct And 'Associated' (after 1976 Indirect and Fortuitous).

Despite the increase in the incidence of operative delivery the number of deaths amongst women delivered by Caesarean section in this triennium has fallen. The breakdown of the numbers and the rate per 1000 Caesarean sections for each triennium is shown in Table 12.3. It can be seen that there has been a 69 per cent fall in the fatality rate since 1970–72.

Table 12.3 *Number of Direct maternal deaths within 42 days of Caesarean section*

	1970–72*	1973–75	1976–78	1979–81	1982–84	Total*
All Direct deaths	343	227	217	178**	138	1103
Direct deaths from Caesarean sections	81	60	61	59	44	305
Percentage of all Direct deaths	24	26	28	33	32	28
Caesarean section rate (%)	5·16	5·63	7·14†	8·90†	10·09	7·31
Estimated fatality rate/1000 Caesarean sections	0·78	0·59	0·51	0·35	0·24	0·45

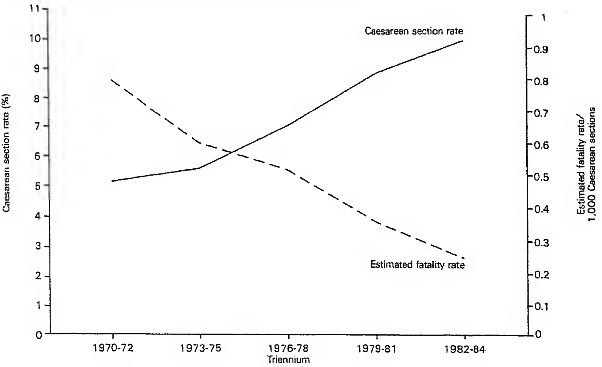
† Revised figures to those previously published; corrected for error in calculation of total maternities in NHS hospitals.

* For changes in numbers and rates since last report see page 153.

**Includes two other Direct deaths omitted from last report.

The Caesarean section rate and estimated fatality rate between 1970 and 1984 is illustrated in Figure 12.1. The proportion of all Direct deaths having a Caesarean section has risen from 24 per cent in 1970–72 to 32 per cent in this triennium.

Figure 12.1 *Caesarean section rate (%) and estimated fatality rate per thousand Caesarean sections. England and Wales, 1970–1984.*



Immediate cause of death

The attributed cause of death amongst the Direct Caesarean section deaths for each triennium since 1970–72 is shown in Table 12.4.

Table 12.4 *The number and the percentage in parentheses of all deaths after Caesarean section classified according to immediate cause of death, 1970–84***

	1970–72**	1973–75	1976–78	1979–81	1982–84
Total	102	77	80	87	69
Haemorrhage	8 (8)	8 (10)	8 (10)	7 (8)	4 (6)
Pulmonary embolus	15 (15)	6 (8)	9 (11)	7 (8)	12 (17)
Sepsis	15 (15)	8 (10)	8 (10)	4 (5)	1 (1)
Hypertensive disease	13 (13)	12 (16)	12 (15)	13 (15)	10 (14)
Anaesthesia	22 (22)	17 (22)	18 (22)	19 (22)	8 (12)
Other Direct causes	8 (8)	9 (12)	6 (8)	9 (10)	9 (13)
Indirect and Fortuitous*	21 (21)	17 (22)	19 (24)	28 (32)	25 (36)

* Referred to as 'Associated Diseases' in previous reports.
**For changes in number and rates since last report see par 153.

There has been a fall in the numbers in each group.

Anaesthesia which was the commonest immediate cause of death in association with Caesarean section is now in third place supplanted by pulmonary embolism and hypertensive disease. Sepsis has shown the most notable fall as an immediate cause of death.

Influence of age and parity

Table 12.5 is a breakdown of the 69 deaths relative to maternal age and parity. It appears that the risk of death increases progressively with age from 25 years, being more than 10 times higher in women over 40 years compared with the rate for women aged less than 25. The mortality rate relative to parity appears to be lowest for para 2 and then increases, being highest for women who have had five or more babies.

Table 12.5 *Number of maternal deaths from Caesarean section (1970–84) related to age and parity*.*

Age in years	Parity					Not stated	Total	Rate/million maternities
	1	2	3	4	5+			
Under 20	22	2	1	0	0	–	25	26·0
20–24	56	16	5	0	0	2	79	25·1
25–29	63	39	14	5	0	–	121	35·9
30–34	33	33	14	12	9	2	103	62·3
35–39	11	10	11	8	14	–	54	102·3
40–44	2	3	6	7	14	–	32	282·6
45 and over	0	1	0	0	0	–	1	104·8
Total	187	104	51	32	37	4	415	
Rate/million maternities	47·9	30·4	34·4	56·8	92·3		42·5	

*For definition of parity see page 154.

Indirect and Fortuitous deaths
(previously 'Associated' deaths)

There were 20 Indirect and 5 Fortuitous deaths. The causes of death are listed below:—

ICD Codes	*Indirect deaths*	
647.6 070.9	Viral hepatitis	1
648+Additional codes:		
153.2	Carcinoma of colon	1
174.9	Carcinoma of breast	2
191.4	Cerebral tumour	1
227.0	Phaeochromocytoma	2
250.1	Diabetes mellitus	1
345.1	Epilepsy	1
410	Coronary atheroma	1
414.1	Coronary artery aneurysm	1
745.4	Eisenmenger's syndrome	1
994.1	Drowning (suicide)	1
997.0	CNS complication resulting from procedure	1
999.3	Transfusion of infected blood	1
674.0	*Cerebrovascular disorders in the puerperium*	
	Cerebral haemorrhage	3
	Cerebral infarction	1
	Carotid artery thrombosis	1
Total		20

ICD Codes	*Fortuitous deaths*	
162.3	Carcinoma of bronchus	1
189.0	Carcinoma of kidney	1
191.6	Cerebellar tumour	1
191.9	Cerebral tumour	2
Total		5

Indications for Caesarean section in Direct maternal deaths

Table 12.6 lists the indications for Caesarean section amongst the 44 Direct deaths relative to whether the operation was elective or emergency and compares the figures with those of 1979–81. The commonest indication was hypertensive disease which in the triennium was also the second commonest cause of death. Other causes are similarly distributed.

Table 12.7 shows, as in the last triennium that, amongst the Direct deaths mortality was highest amongst women undergoing emergency Caesarean section compared with those having the elective operation, although deaths amongst this group fell more than amongst the elective. The reasons for the persistence of a higher mortality rate amongst the emergency group is not certain. Undoubtedly the conditions for which this operation is performed carry a greater risk amongst women undergoing an emergency Caesarean section. However it is also likely that this emergency operation is inherently more dangerous because of the lack of preparation and the stressed condition of the mother. In this country, most emergency Caesarean sections are done by junior obstetricians and anaesthetists and the risk of the operation can be reduced only by full consultation with, and backup from, their seniors. Experience shows that it is rarely possible to predict the need for delivery by emergency Caesarean section and it is likely that any attempt to reduce the number by performing more elective operations would simply increase the overall Caesarean section rate.

Table 12.6 *Indications for Caesarean sections among Direct maternal deaths. Figures in parentheses are the percentages from previous triennium (1979–81).*

	Numbers from		Total	Percentages
	Elective Caesarean	Emergency Caesarean		
Hypertensive disease of pregnancy	1	13	14	32 (30)
Fetal distress	1	5	6	14 (24)
Delay or failure to progress in labour	0	8	8	18 (17)
Antepartum haemorrhage	1	5	6	14 (12)
Maternal conditions	3	3	6	14 (7)
Previous Caesarean section	2	1	3	7 (7)
Malpresentation	0	1	1	2 (3)
Total	8	36	44	101 (100)

Table 12.7 *Number of Direct and Indirect deaths of women delivered by Caesarean section and rates related to whether an emergency or elective operation was performed.*

	Estimated total number Caesarean sections			Number of deaths and rate (per 1000 operations)	
	1979–81	1982–84*		1979–81	1982–84
Elective	85,000	86,000	Direct deaths	9 (0·11)	8 (0·09)
			Indirect deaths	11 (0·13)	11 (0·13)
			Total	20 (0·24)	19 (0·22)
Emergency	81,000	101,000	Direct deaths	50 (0·62)	36 (0·36)
			Indirect deaths	17 (0·21)	14 (0·14)
			Total	67 (0·83)	50 (0·50)

*Figures for Caesarean sections in NHS hospitals in England in 1982–84 are available from HIPE. These figures are subdivided into the categories elective and emergency. Figures for Caesarean sections in NHS hospitals in Wales in 1982–84 are available from the Welsh HAA. These figures cannot be subdivided into elective and emergency procedures. For the purposes of Table 12.7 the split between elective and emergency procedures in Wales was assumed to be in the same proportions as that of England.

Postmortem Caesarean sections

Table 12.8 shows details of five women and their babies on whom Caesarean sections were carried out after death of the mother. As in previous triennia the cases reveal how difficult it is to rescue a baby once the mother has died. The only exception is the woman who died from an amniotic fluid embolus whose baby survived. This may well be due to the acute nature of the death of a woman whose baby is likely to be in good condition before the embolism. However it is difficult to be selective at the time of death and despite the risk of delivering a baby that survives in a brain-damaged condition, obstetricians have no option but to make the attempt.

Table 12.8 *Cause of death in women delivered by postmortem Caesarean section*

ICD Code	Cause	Outcome for baby
Direct		
673.1	Amniotic fluid embolus	Survived
Indirect		
674.0	Subarachnoid haemorrhage	Stillbirth
648.6, 441.0	Dissecting aortic aneurysm	Early neonatal death
648.9, 345.3	Epilepsy	Stillbirth
Fortuitous		
320.9	Meningitis	Early neonatal death

Substandard obstetric care

The death of a woman delivered by Caesarean section is often not primarily caused by the operation itself. In this triennium, only 12 of the 69 women died from haemorrhage or complications of anaesthesia. For this reason, only Direct deaths have been considered in the assessment of whether care was substandard or not, and this assessment relates to the total care provided before, during, and after the operation.

There was sufficient evidence in 41 of the 44 Direct deaths to show that care was substandard in 23 of them.

Table 12.9 summarises major factors contributing to substandard care. The most frequent single factor was inadequate control of hypertension. Faulty management of complications immediately before and after the operation was the next most common. Delay in deciding to perform a Caesarean section or in transferring a woman from a GP unit was a factor in three cases. Junior staff were asked to accept responsibility beyond their capabilities in three cases. Three women went against medical advice by leaving hospital or refusing treatment, which was a major contributing factor to their deaths.

In 11 cases, problems of management contributed to the deaths that were the responsibility of the consultant obstetrician. In 5 cases the obstetric registrar or senior registrar was responsible, and in one, the obstetric senior house officer.

Table 12.9 *Cases of substandard care classified according to major factors contributing to Direct deaths from Caesarean section*

1. Anaesthetic/resuscitation		9
2. Inadequate control of hypertension		4
3. Operation done too late		3
4. Poor management of haemorrhage		1
5. Undue responsibility placed on junior staff		3
6. Poor management		6
—antenatal preparation	2	
—pulmonary embolism	1	
—deep vein thrombosis	1	
—fluid balance	2	
7. Patient non-collaboration		3

A case history is given as an example of a combination of non-cooperation from the patient, and substandard medical care contributing to the death. A woman from a good background had an uneventful second pregnancy until the end of the second trimester, when she developed signs of pre-eclampsia. She declined to come into hospital, despite the advice of the obstetrician, because she wished to complete her job before starting maternity leave a week later. She had an eclamptic fit six days later and was taken to hospital. She had a second fit whilst being transported and a further fit in hospital. A Caesarean section was done but during the operation she became severely hypertensive. Despite good control of blood pressure postoperatively she had a further two fits when she was first seen by the consultant obstetrician more than eight hours after admission. One hour later she was pronounced brain dead.

This case illustrates three important points. The first is the common problem encountered nowadays of convincing women who have a potentially dangerous complication of pregnancy of the need to give up work when they are feeling well. The second is the importance of prescribing anticonvulsant therapy in addition to controlling the blood pressure in women who have eclampsia or fulminating pre-eclampsia. The third is the need for the most senior member of the obstetric team to see women with potentially life-threatening complications as soon as possible after they come into hospital.

Substandard anaesthetic care

There were eight women delivered by Caesarean section and three women who were being prepared for Caesarean section whose deaths are counted in Chapter 13. Nine of them were found to have had substandard care, seven with substandard anaesthesia alone, and two having both obstetric and anaesthetic care which was substandard.

Nine had a general anaesthetic and in seven there was difficulty at the time of intubation or aspiration of gastric contents. The other two died from inadequate reversal of curarisation.

Anaesthesia continues to be a major hazard, and although the number of cases is relatively small, there are still lessons to be learnt. Early involvement of the duty anaesthetist in any case where operative delivery is more than just a possibility, particularly in very obese or short women, or in those with a past history of difficult intubation, allows junior anaesthetists to discuss the case with a senior. The absence of any deaths in which epidural anaesthesia has been implicated although it is likely that the cases for which it was used were different from those who had a general anaesthetic, suggests that, where possible, it is the anaesthetic of choice.

Conclusions

The most remarkable feature of the information presented in this chapter is the continued decline in maternal deaths despite increasing numbers of Caesarean sections shown in Figure 12.1. It seems likely that the reason for this is improved clinical care, better patient selection, better anaesthesia and better surgical technique.

The fact that in many cases care was judged to have been substandard shows that there is room for improvement. The quality of management of acute hypertension is still very variable, and proposals for this are made in Chapter 2. Junior medical staff continue to be expected to carry too great a responsibility in the labour ward, and consultants need to be prepared to come in earlier to see difficult cases. General anaesthesia is, as always, a major hazard and although the number of cases is relatively small there are special lessons to be learnt. Early involvement of the duty anaesthetist in any case when operative delivery is more than just a possibility, particularly in very obese or short women or those with a past history of difficult intubation, allows junior anaesthetists to discuss the case with a senior. The absence of any deaths in which epidural anaesthesia has been implicated suggests that, where possible, it is preferable to general anaesthesia.

The overall picture is one of improving safety of Caesarean section in the face of an increasing number of operations being done. These good figures should not be seen as a reason for disregarding the accepted policy of only resorting to Caesarean section when other methods are considered, for whatever reason, to be inappropriate. Delivery by Caesarean section, particularly if a general rather than an epidural anaesthetic is used, remains significantly more hazardous than vaginal delivery.

13. Maternal deaths associated with anaesthesia

Summary

Nineteen deaths were associated with anaesthesia in this triennium, 18 of them directly due to anaesthesia and one in which anaesthesia contributed to the death. Seven deaths were due to the sequelae of the inhalation of stomach contents into the lungs. Two of these were primarily due to difficulty with endotracheal intubation which accounted for 10 deaths altogether, the other eight dying from the effects of anoxia. One of these cases does not appear in the total; being a late death, it is counted in Chapter 16. In five patients post-operative care was deficient and haemorrhage played a part in the deaths of four patients. One patient died from the misuse of drugs, one from misuse of apparatus, and one from a total subarachnoid block complicating epidural analgesia.

In this report, the commonest aspect of substandard care was the inexperience of the anaesthetist, and it is our view that, in most instances, this was a failure in hospital administration. There has been a slow improvement in the anaesthetic mortality figures over the past six triennia, but it is still evident that some pregnant women are not accorded the standard of care that is generally available to surgical patients, particularly with respect to the facilities, equipment, and the supervision of staff in training.

Maternal deaths associated with anaesthesia

Nineteen deaths were associated with anaesthesia in the years 1982–84, 18 directly due to anaesthesia and one in which there was an anaesthetic contribution. In addition there was one direct death in 1983, which was a late death, and is counted in Chapter 16. There was also one case, discussed in the chapter on haemorrhage, which is of interest from the anaesthetist's point of view and in which there was a possible anaesthetic contribution.

Deaths have again been classified according to ICD 9 under code 668, 'Complication of the administration of anaesthetic or other sedation in labour and delivery'. Deaths have been grouped into sections under the headings of the main anaesthetic causes. The 'Miscellaneous' section varies with each report.

As is now customary, deaths either directly due to anaesthesia or in which anaesthesia played a significant part are classified according to the initiating anaesthetic factors. The figures cannot be statistically significant in the sense of trend prediction from report to report since the number of women anaesthetised without mishap for any given obstetric condition cannot be determined.

As for past Triennial reports, readers should be aware that the MCW97 forms are carefully analysed by the central and regional assessors and a 'best fit' diagnosis of the cause of death is used. It is fairly certain to be correct in most cases, but where there is doubt about the sequence of events, this is discussed in the text. In this chapter and throughout the report, the term 'substandard care' has been used instead of 'avoidable factors' since it reflects better the circumstances of the deaths and is less pejorative.

Table 13.1 Deaths, associated with anaesthesia, estimated rate per million pregnancies and percentage of Direct maternal deaths 1970–84*

	1970–72*	1973–75*	1976–78*	1979–81	1982–84	1970–84
Number of deaths directly associated with anaesthesia	37	27	27	22	18	131
Rate per million pregnancies	12·8	10·5	12·1	8·7	7·2	10·2
Percentage of Direct maternal deaths	10·8	11·9	12·4	12·4	13·0	11·9

*For changes in numbers and rates since last report see page 153.

Table 13.2 Deaths associated with anaesthesia: procedures for which anaesthetic was given

Operation	Indication	Number of Deaths
Laparotomy	Ruptured ectopic pregnancy	3
Dilatation and curettage with tubal ligation	Legal termination with sterilization	1
Proposed evacuation of retained products of conception (Died during induction of anaesthetic)	Incomplete abortion	1
Forceps delivery	Delay in 2nd stage of labour	1
Proposed emergency Caesarean section—vaginal delivery	Fetal distress	1
Proposed elective Caesarean section (Died during induction of anaesthetic)	Fetal growth retardation	1
Elective Caesarean section	2 Previous Caesarean sections	1
Proposed emergency Caesarean section (Died during induction of anaesthetic)	Failed forceps delivery	1
Emergency Caesarean section	i. Pre-eclampsia	1
	ii. Failed forceps delivery	1
	iii. Breech presentation	1 }7
	iv. Fetal distress	1
	v. Prolonged labour	3
Manual removal of placenta	Retained placenta	1

Table 13.1 gives the Direct deaths from anaesthesia, as an estimated rate per million pregnancies and as the percentage of all Direct maternal deaths from 1970 to 1984. The trend has been towards a decline in numbers with a marked fall in the death rate per million. The improvement is probably greater than the figures indicate because the number of anaesthetics given to pregnant women for abortions and operative delivery has increased during this period.

Table 13.2 deals with Direct deaths from anaesthesia showing the procedures for which the anaesthetic was given. Fourteen of the anaesthetics were for emergency and four for elective procedures. This does not differ significantly from the last triennium, when 15 deaths occurred in patients having emergency Caesarean section with four in elective cases.

Inhalation of stomach contents

In this triennium, the deaths of seven patients were due to the sequelae of the aspiration of stomach contents into the lungs. Aspiration was associated with difficulty in endotracheal intubation in two, with deficient post-operative care in three and was complicated by haemorrhage in one. In all seven patients, care by the anaesthetists was judged to have been substandard, although in some cases, there were contributions by the obstetric and nursing teams.

Two cases presented complicated pictures which were difficult to interpret. One grossly obese woman had considerable blood loss due to a retained placenta following spontaneous delivery after a prolonged labour. Anaesthesia for manual removal was induced intravenously, followed by nitrous oxide and oxygen with 2 per cent halothane by mask and oro-pharyngeal airway. A clear picture of aspiration pneumonitis developed shortly after operation, heralded by progressive cyanosis and tachycardia and with severe acidosis and a large base deficit. There had been inadequate blood and fluid replacement, but the chest radiograph was interpreted as pulmonary oedema due to fluid overload although there was no renal response to intravenous diuretics. Death was preceded by disseminated intravascular coagulation. The case is mentioned in Chapter 3. This responsibility was much too great for the junior anaesthetist who did not recognise the risks and who lacked the expertise to deal with the case. The other case was of a patient who had had an attempted forceps delivery followed by Caesarean section under general anaesthesia. Although there was no reported anaesthetic mishap or overtly unacceptable management, the patient developed some respiratory difficulty at the end of operation, which progressed to the adult respiratory distress syndrome, and she died a week later. Autopsy revealed no evidence of amniotic fluid embolism.

Of interest is one patient with a ruptured ectopic pregnancy for which a salpingectomy was performed. She was then returned to a general ward in the middle of the night still drowsy and heavily sedated, where she regurgitated and inhaled stomach contents. Post-operative restlessness had been treated with additional narcotic although it was probably due to hypoxia.

Another patient had an uneventful elective Caesarean section but aspirated stomach contents either at the end of operation, the most probable event, or shortly thereafter in the ward. She required intensive care within two hours of surgery and died from respiratory failure three days later. The other death from aspiration was that of a woman who was admitted some weeks before delivery for bed rest for hypertension and proteinuria. Labour was induced with a prostaglandin pessary because of some elevation of blood pressure but next day her blood pressure rose precipitately and she was treated with diazepam and taken to theatre for Caesarean section. She regurgitated a large volume of fluid with aspiration following the administration of suxamethonium, there being no cricoid pressure. Tracheo-bronchial suction removed some of the fluid, hydrocortisone was given and the operation proceeded. She was returned to the ward without further attention. She soon developed severe respiratory distress and died 10 days later in the intensive care unit. The junior anaesthetist did not appreciate the seriousness of inhalation of stomach contents although it is uncertain whether immediate post-operative admission to the intensive care unit would have influenced the outcome.

Anaesthetists in general are now very much aware of the dangers of aspiration pneumonitis in obstetric practice, but the anaesthetists concerned were not skilled enough to avoid these casualties.

Difficulty with endotracheal intubation

There were 10 deaths primarily due to difficulty with endotracheal intubation, and in addition, a late death counted in Chapter 16.

Of these 10 patients, two died from the pulmonary effects of aspiration of stomach contents which occurred during attempts at intubation, eight from anoxic cardiac arrest, from brain damage caused by an arrest, or primarily from anoxic brain damage.

The histories of the two patients who died from the effects of aspiration of stomach contents are of interest. One, who had severe pre-eclamptic toxaemia was small in stature, very obese, and with a short fat neck. Having failed to progress in labour, she was anaesthetised by an inexperienced anaesthetist for an emergency Caesarean section. The initial attempt at intubation was followed by severe bronchospasm, presumably due to aspiration and this proceeded to anoxic cardiac arrest with brain damage from which she died some days later. The difficulty had been anticipated and discussed with senior staff, but anaesthesia and surgery were left to juniors. In the face of laryngospasm and bronchospasm, the failed intubation drill was unhelpful.

The second death from aspiration was of a small physically abnormal, subfertile woman with a protruding jaw. A trial of labour was ended when fetal distress occurred and she was delivered by Caesarean section. The anaesthetist was unable to intubate the patient and severe respiratory distress from aspiration of gastric contents was evident at the end of operation. She died within a few hours. Anaesthesia was the primary factor in her death since the anaesthetist maintained anaesthesia for Caesarean section with nitrous oxide and oxygen and intermittent doses of suxamethonium, ventilating the lungs by bag-squeezing and mask. Other aspects of her care were substandard. There was no consultation with the anaesthetists. With all the adverse obstetric factors it seemed unwise to embark on a trial of labour which was likely to end in emergency Caesarean section. The autopsy showed some evidence of early viral pneumonia and marked congenital narrowing of the glottis. The on-call anaesthetist had to manage this difficult case as an emergency which greatly lessened her chance of survival.

Of the other eight patients, one was given a general anaesthetic for elective Caesarean section by an experienced trainee anaesthetist, following the failure of an epidural block. He failed to intubate and the patient was in extremis from hypoxia before the consultant who was called made further attempts. Finally an attempt at tracheostomy by the consultant also failed and the patient died within a few minutes. Autopsy showed that this small woman had an abnormally small larynx and trachea and that the cause of death was simple anoxia.

One patient died undelivered during anaesthesia for elective Caesarean section, as the result of an overinflated cuff which obstructed the lumen of the endotracheal tube. The patient died of anoxia. In another case, an endotracheal tube became kinked in the pharynx and obstructed when the patient, being anaesthetised for a therapeutic abortion and sterilisation, was left in the care of an operating department assistant. The East Radcliffe ventilator also became disconnected from the fresh gas supply although this was secondary in importance to the obstruction of the tube. The patient died a few days later from the effects of cerebral anoxia.

In another case, an inexperienced anaesthetist was called for an emergency Caesarean section. He passed an endotracheal tube with difficulty and removed it for further intubation attempts only after cardiac arrest had occurred. Differing views were held about the primary factors in this case, including a possible allergic reaction to methohexitone or suxamethonium, but to the assessors it seemed to be most typical of an oesophageal intubation which was maintained until it was too late for resuscitation to succeed.

Another patient died on the operating table due to difficulty with endotracheal intubation. She was undergoing general anaesthesia for an emergency Caesarean section. The registrar anaesthetist could not intubate the patient who was obese, with a short neck, and a small larynx which could not be seen on laryngoscopy. Once the effect of suxamethonium had worn off, attempts to do a tracheostomy were made, but an airway could not be maintained. This caused haemorrhage into the trachea and lungs which rapidly filled with blood, and despite help from a consultant who was available within a few minutes, all attempts at resuscitation failed and the patient died undelivered.

Another woman was conventionally anaesthetised without difficulty by face mask, for a curettage, following a suspected incomplete abortion. The anaesthetist passed an endotracheal tube when laparoscopy was proposed for a probable ectopic pregnancy. The story is then vague and confused, but on balance, the best explanation is that the oesophagus was intubated and death occurred from brain damage due to anoxia. It was unfortunate that the tube was not removed when it disrupted an otherwise uneventful anaesthetic.

One other patient suffered from progressive cyanosis which started soon after intubation, while she was being anaesthetised for removal of retained products of conception. Her pre-operative condition was excellent but she must have presented some anaesthetic difficulty because she was obese. Cardiac arrest occurred within ten minutes of starting anaesthesia and she suffered lethal brain damage. This also was due to oesophageal intubation.

The last patient in this section suffered severe bradycardia and probable cardiac arrest during attempted intubation by an inexperienced anaesthetist. The patient had been admitted semi-comatose and in severe hypovolaemic shock from a ruptured ectopic pregnancy. Pre-operative management was very poor and no senior anaesthetist was concerned with the case until cardiac arrest occurred. The endotracheal tube was placed in the oesophagus at the first attempt and she was not in good enough condition to withstand the period of hypoxia before the anaesthetist recognised his error and corrected it. Blood was not given until surgery was under way and the blood loss was clearly the major factor in this death which is counted in Chapter 5.

The late death occurred three months after delivery as a result of hypoxic brain damage which occurred during difficulty with endotracheal intubation. The patient was grossly obese and was admitted for elective Caesarean section because of hypertension and proteinuria. She had a short and very thick neck and the consultant working without skilled assistance failed to intubate the trachea. A tracheostomy was performed by the surgeon in an attempt to relieve the progressive hypoxia which followed and which led to cardiac arrest. External and then internal cardiac massage together with other conventional measures restored the heartbeat, but the patient did not regain consciousness and entered a vegetative state, dying some three months later from pneumonia.

In all but one of these deaths primarily due to intubation problems, anaesthesia was given by unsupervised, inexperienced, trainee anaesthetists and this constitutes substandard care.

There is a need for more balanced teaching of juniors about the problems of intubation and in particular, the anaesthetists and obstetricians in every obstetric unit should practice failed intubation drills. There is no evidence that any death could have been prevented had the anaesthetic been given by a consultant but some would have been better managed by prior discussion between the obstetrician and anaesthetist.

Failure of post-operative care

Five patients died whose post-operative care was substandard. Three died from the inhalation of stomach contents and have been described under that heading. One died from the unskilled management of artificial ventilation in the ward and is described under 'Misuse of Drugs'. The last one had a cardiac arrest in the ward due to respiratory obstruction and hypoxia from the inefficient reversal of tubocurarine at the end of an emergency Caesarean section. The atropine (1·2 mg) and neostigmine in two doses of 2·5 mg, were lost into the tissues through displacement of the intravenous cannula. Recovery facilities and observation in the ward were inadequate and it was substandard care for this patient to be returned to the ward, unconscious and with an observed and noted tracheal tug, indicative of intercostal paralysis from persistent neuromuscular blockade.

Haemorrhage

Haemorrhage played a part in the deaths of four patients. Two of these are described in the paragraph on difficulties with endotracheal intubation. Another one died from aspiration, pneumonitis but haemorrhage complicated the picture and the case is mentioned but not described in Chapter 3. Blood loss was inadequately managed by the anaesthetist. This patient with the retained placenta is described in the paragraph on inhalation of stomach contents. Her post-operative haemoglobin value of just over 5g/dl must have contributed to her demise. The last one already described, virtually drowned in blood from an attempted tracheostomy.

Another complicated case is counted in Chapter 3. This patient died following an elective Caesarean section complicated by post-partum haemorrhage for which two laparotomies were performed. She died from disseminated intravascular coagulation. Anaesthesia could possibly have played a part in its onset in that she developed some bronchospasm following induction of anaesthesia and endotracheal intubation. This responded rapidly to aminophylline and corticosteroid, and operation proceeded without hindrance. She was massively obese and a known asthmatic, in most competent anaesthetic care.

Misuse of drugs

Only one death is classified as being due to the misuse of drugs. A multipara had labour induced with prostaglandin pessaries, but because of fetal heart deterioration, it was decided to proceed to Caesarean section. However, once anaesthetised the patient delivered spontaneously. The anaesthetist was inexperienced and used pancuronium for neuromuscular block, the effect of which could not be reversed at the end of this short procedure and, as there was no intensive care unit in the hospital, he left the patient connected to a Manley ventilator in the ward, in the care of nurses who had inadequate experience of artificial ventilation. The patient developed severe hypoxia and hypercarbia due to ventilation deficiency and died from cardiac arrest. Treatment once her condition was discovered was inappropriate and she died without recovery of consciousness. Care was substandard in the choice of neuromuscular blocking drug and in the post operative management.

Pancuronium cannot now be recommended for use in operative obstetrics because of its long duration of action and the difficulty of early reversal of its effects. There are now many safe alternatives with appropriate durations of action.

Miscellaneous

There was one death associated with the misuse of anaesthetic apparatus. The patient was undergoing laparotomy for a ruptured ectopic pregnancy when hypoxia developed which progressed to cause cardiac arrest and death from brain damage three weeks later. The anaesthetist had failed to connect the oxygen supply properly to the East Radcliffe ventilator. From many reports in the literature it seems that this type of machine is unsafe for routine use in anaesthesia without the monitoring of the inspired oxygen concentration. It is not fail-safe and can be misused as in this case.

One death occurred in association with epidural analgesia. It seems clearly to be a case where the administrative arrangements of the unit were at fault. An epidural block with bupivacaine was instituted by a junior anaesthetist and lasted for some five hours. Top-up was delayed until a decision to do a forceps delivery had been made and the dose was administered by a midwife, there being no anaesthetist available in this isolated maternity unit. The midwife was called away shortly after doing so and on her return the patient was noted to be blue. Total spinal block was present and cardiac arrest followed. During attempts at resuscitation, the endotracheal tube was misplaced and efficient ventilation was not established before lethal anoxic brain damage had occurred.

The primary failure of care lay in the lack of continued observation following the top up dose of bupivacaine, because total subarachnoid block should not be lethal. The Standing Advisory Committee on Obstetric Anaesthesia and Analgesia, a joint committee of the Faculty of Anaesthetists of the Royal College of Surgeons of England and the Royal College of Obstetricians and Gynaecologists, recommend that 'in the interests of patient safety, a person proficient in the cardio-pulmonary resuscitation of a pregnant woman should be available within the maternity unit at all times if an epidural is in place'. This policy has been accepted by both bodies.

The last patient was admitted in her first pregnancy with severe pre-eclamptic toxaemia and had an emergency Caesarean section. She had severe osteogenesis imperfecta with marked kyphoscoliosis, and although anaesthesia did not contribute directly to her death, the limited experience of the aneasthetist did not provide her with the best chance of survival. Over the years she had suffered from multiple fractures and had been confined to a wheelchair for about ten years. She concealed her pregnancy until admission for severe hypertension, oedema and albuminuria. The patient died as the result of gross pulmonary oedema following multiple transfusions for post-operative bleeding.

Pathology

The standard of anatomical autopsy report in 15 of the 18 Direct deaths described in this chapter was above average. However in only eight (44 per cent) was an adequate histological report included. In the two cases in which there was need, appropriate special investigations were done.

Conclusions

The number of deaths associated with anaesthesia has diminished fairly steadily over successive triennial periods and since 1964 has been 50, 50, 37, 30, 28, 22 and now 19. This decline in numbers is likely to represent a true reduction in the incidence of death associated with anaesthesia because it is fairly certain that there has been a progressive increase in the numbers of anaesthetics administered to women in pregnancy, labour and the puerperium since 1964. The reduction is probably related to the growing awareness in the National Health Service over this period that obstetric anaesthesia cannot safely be left to unsupervised beginners in the speciality, and that the woman in labour has to have available to her, all the anaesthetic resources and skills which are provided for patients having other surgical procedures. Black spots however remain in the system as this report shows.

Care was substandard in all of the deaths directly due to anaesthesia. Failure to intubate satisfactorily was the primary cause in 10 and reflected the administrative failure of leaving inexperienced junior anaesthetists to care for patients who required the most skilled attention. There was evidence of poor liaison between the obstetric and the anaesthetic teams and deaths might have been averted in some instances if there had been acceptable cooperation. Obstetricians must also to some extent share in the responsibility for their acceptance of the staffing of units by inappropriately experienced anaesthetists.

Some of the case histories indicate that there is a belief that Caesarean section cannot be carried out in the absence of full muscular relaxation produced by neuromuscular blocking drugs. This is not the case. All that is required is light general anaesthesia, (Guedel Stage 3 Plane (i)), because the delivery of the baby and the contraction of the uterus allow easy intra-abdominal manipulation and closure of the abdominal wall. General anaesthesia of this depth maintains the laryngeal reflexes, providing protection against the inhalation of vomitus. Passive regurgitation from the stomach is also unlikely at this anaesthetic depth, so that light general anaesthesia with a volatile agent administered by mask and oro-pharyngeal airway, as part of a failed intubation drill is not as fraught with hazards as seems to be generally assumed.

Some of the case histories also disclose a belief that volatile anaesthetics are inimical to the successful resuscitation of the baby, a belief supported in the literature on anaesthesia by many reports of awareness during Caesarean section under nitrous oxide analgesia with neuromuscular block. In theory, volatile anaesthetics given to the mother might depress the baby and lower the Apgar score at birth, but there is no acceptable evidence that low blood concentrations of volatile general anaesthetics in the baby are in any practical way harmful, even in cases of fetal distress. In the short period between induction of anaesthesia and delivery, blood concentrations in the fetus are unlikely to reach any significant level. It is suggested that the old skill of rapidly producing and maintaining light general anaesthesia without the use of neuromuscular blocking drugs or intravenous induction agents should be revived, so that the technique could be employed with confidence when things go wrong.

In this report, six of the women dying from aspiration of stomach contents had the recommended dosage regimen of magnesium trisilicate mixture to raise the intra-gastric pH during labour and before delivery. The 7th, with a ruptured ectopic pregnancy did not have antacids. These deaths can be described as being due only to aspiration of stomach contents since the nature of the inhaled material was not determined. The prevention of death and morbidity from aspiration is already the subject of much ongoing research and the literature on the subject is extensive. A major part of the research has been into methods of raising intra-gastric pH during labour since it is accepted that it is desirable to keep it above 2·5, a level shown by experiment to be the threshold for the generation of the acid aspiration syndrome. In the light of the cases in this and previous reports, one must again question whether the administration of substantial volumes of a particulate antacid is safe. It seems that in the reported cases it was either ineffective in raising the pH, or the inhalation of the antacid itself resulted in lethal lung damage. In the light of the very small numbers of deaths it will never be possible to mount a controlled trial of alternative therapies in the prevention of death from aspiration in obstetric patients. However, it might well be, that the intra-gastric pH can be better controlled by H2 receptor antagonists and sodium citrate combinations. It is equally essential not to lose sight of the importance of trying to reduce the volume of the stomach contents during labour and methods of acid control should not prejudice this.

It is almost universally held that the stomach does not empty in normal labour. While there is no evidence that this is the case, there is ample evidence that the use of narcotics, most commonly the use of pethidine, inhibits gastric motility and prevents emptying of the stomach for many hours following even a single analgesic dose. The almost universal use of narcotics should be re-examined because their omission would be a contribution to patient safety.

Finally, the practice of endotracheal intubation is a basic skill required of all anaesthetists, yet difficulty with it was the commonest anaesthetic factor leading to death in this triennium. One can only surmise that the skill necessary to deal with the really difficult intubation is only acquired after some years of experience, principally because the great majority of intubations in non-pregnant surgical patients are easy, even for the beginner. In 'surgical' anaesthetic practice, good pre-operative assessment and planning, which are the keys to safety, are generally adopted, but are sometimes omitted in obstetric practice for a variety of reasons which are apparent from this and previous reports. Patients at particular risk from failure to intubate are women of short stature who are obese with short, fat necks. This combination of physical features should alert the obstetrician and midwife to the need for consultation with the anaesthetist. Failure to intubate the properly prepared non-pregnant patient is much less likely to result in morbidity than in the patient in labour.

Inexpertly applied cricoid pressure can make intubation very difficult or even impossible and this may well account for some of the failures. Pressure on the cricoid cartilage can cause flexion of the head on the neck and prevent visualisation of the larynx. The Sellick manoeuvre requires the use of two hands, one to press on the cricoid cartilage, and the other to maintain the head in the proper attitude of neck flexion with extension at the atlanto-occipital joint. The technique should be practised on surgical patients, particularly in obese, short-necked females, so that it can be perfected without the dangers of trying to do so in the middle of the night in emergency obstetric cases. A trained assistant is absolutely essential to ensure that it is done properly and also to take part in the failed intubation routine, should this become necessary.

14. Indirect causes of maternal death

Summary

There were 71 Indirect deaths representing 34 per cent of all Direct and Indirect deaths, a similar proportion to the last triennium. Fourteen were deaths from cardiac disease, described in Chapter 10; the remaining 57 deaths are discussed in this chapter.

Classification and presentation of cases*

Indirect maternal deaths are defined as those resulting from a previously existing disease, or disease that developed during pregnancy and which did not have a direct obstetric cause but which was aggravated by the physiological effects of pregnancy. This definition has also been interpreted here to include deaths in which the presence of the existing disease resulted in significant changes in the treatment or management of the pregnancy, or of the condition itself, eg diabetes, epilepsy.

To reflect the increased importance and interest of these 57 deaths, more detailed descriptions of some of the cases and their treatment have been given in this report than previously, because of the possibility that with a better understanding of conditions, such as epilepsy, death can be prevented. Some conditions previously considered as Fortuitous have been reclassified in the light of recent evidence as Indirect. This continues the trend begun in the 1979–81 report, when all deaths from intracranial haemorrhage were for the first time classified as Indirect.

Substandard care

In 10 of the 57 cases care was regarded as substandard.

Infectious diseases

Septicaemia

There were three deaths from septicaemia.

The first was a primigravida, whose pregnancy had proceeded entirely normally until 37 weeks gestation when she developed acute tonsillitis for which her general practitioner prescribed a linctus. One week later she was admitted to hospital with an acute febrile illness and an intrauterine death. She rapidly developed acute disseminated intravascular coagulation (DIC) due to a beta-haemolytic streptococcal infection. She died despite appropriate treatment.

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*In this report Fortuitous deaths have been listed in Chapter 16.

The second case involved a multipara whose pregnancy was uneventful until 35 weeks when she developed acute tonsillitis for which she was treated with anti-pyretic drugs by her general practitioner. Ten days later she was admitted to hospital with acute septicaemia and DIC due to a *beta-haemolytic streptococcus*. Despite all efforts she died seven hours after admission.

These two cases illustrate the virulence of *beta-haemolytic streptococci*. Acute tonsillitis or pharyngitis in pregnancy is always potentially dangerous and it is wise to take immediate throat swabs. If a *beta-haemolytic streptococcus*, Lancefield Group A, is isolated the patient should at once be treated with antibiotics, of which penicillin is still the first choice.

The third patient was also a multipara. At 38 weeks gestation she was admitted to hospital in good health for an elective Caesarean section after an uneventful pregnancy. Although the operation was uncomplicated the patient was transfused with two units of blood. She developed acute hepatic failure within 24 hours of delivery and was transferred to an ICU but died five days later. A careful and detailed autopsy revealed massive hepatic necrosis. Investigation of the transfused blood and virological studies of the patient failed to reveal any causative agent. A viral infection, however, was thought to be the most likely cause. Management of this case was at all times of a high standard.

Neoplastic diseases

There were 11 deaths associated with neoplastic disease.

Table 14.1 *The sites of the tumours in deaths from neoplasms*

Site of tumour (benign and malignant)	ICD number	Number of patients	Age at death (years)
ICD Code 648 with additional code:			
Benign			
Glioma	191.4	1	20
Phaeochromocytoma	227.0	4	22;26;31;36
Malignant			
Carcinoma of colon	153.2	1	24
Carcinoma of breast	174.9	5	29;31;36;38;41

It is of interest to note that in two of the four cases of phaechromocytoma the diagnosis was not considered although one of these women had multiple neurofibromata. In another case, characteristic symptoms were noted pre-conception but investigations were not carried out during pregnancy and the last patient had normal catecholamine levels when hypertension was investigated early in pregnancy.

Three of the five patients who had carcinoma diagnosed during pregnancy were offered termination of pregnancy but refused. One patient had breast carcinoma treated prior to her pregnancy and was advised that there was no contraindication to pregnancy. The triennial reports do not allow a detailed analysis of the risks but each report contains at least one case of extensive metastases appearing during pregnancy in patients in whom breast carcinoma appeared to have been eradicated.

At the present time, there is no way of predicting which patients are at risk and which are not of developing metastases. There does not appear to be a consensus opinion from the experts in this field we have consulted as to the most appropriate pre-pregnancy counselling advice.

Endocrine, nutritional and metabolic disorders and immunity disorders

i. *Diabetes mellitus*

Two women with insulin-dependent diabetes died from complications of the disease, and in one diabetic woman, the cause of death was uncertain.

The first death was that of a young woman who had juvenile onset diabetes of twenty years duration. She had had one pregnancy previously which had ended at 33 weeks with the delivery of a macerated stillbirth. She was known to have severe complications of diabetes with nephropathy and retinitis, and had been repeatedly advised against pregnancy, and had refused termination of this pregnancy. Extremely close supervision was provided by both obstetricians and physicians. Early delivery at 28 weeks had been considered but had been deferred because of a low amniotic fluid lecithin-sphyngomyelin ratio. At 33 weeks gestation she was transferred to the regional obstetric unit with severe oedema of the lower limbs of renal origin, and mild left ventricular failure. Ten days after transfer she was delivered of a healthy baby by Caesarean section which was uncomplicated. However, 18 hours after delivery she had a myocardial infarct. From that time onwards her condition slowly deteriorated, and she died 35 days after delivery. Autopsy revealed advanced atherosclerosis with severe stenosis of the coronary, left renal and left carotid arteries. There was also evidence of renal, pituitary and myocardial infarction.

The second woman, para 2, had been a diabetic for nine years. Her two pregnancies 8 and 10 years previously had been entirely normal, presumably because at that time her diabetes was mild. In the first trimester of this pregnancy she developed acute pyelonephritis which destabilised her diabetes, and good control was never subsequently achieved. She died in severe ketoacidosis at 28 weeks gestation. A careful autopsy with histology revealed no significant morphological abnormalities and this must be regarded as a biochemical death. Such cases must be extremely rare, but the dangers of uncontrollable ketoacidosis in pregnancy are so great that consideration should be given to terminating the pregnancy, when it occurs.

The third patient was an insulin dependent diabetic who was mentally backward and suffered from epilepsy. Pregnancy was not recognised prior to her death. The cause of death was uncertain, but she died after collapsing and hitting her head. The autopsy revealed advanced coronary atheroma with associated cardiac ischaemia, with no evidence of a skull fracture or injury to the brain. The uterus was enlarged and contained a fetus of estimated gestational age of 14 to 16 weeks.

ii. *Auto-immune disease*

This case involved a young woman with a history of a legal abortion followed by a spontaneous and then a missed abortion and finally a delivery at 36 weeks gestation of a growth retarded fetus. The last two pregnancies had been complicated by fulminating pre-eclampsia. The current pregnancy had progressed normally until 20 weeks when multiple abnormalities of the fetus were detected and the woman was admitted for a legal abortion by hysterotomy. The operation was uneventful, but 45 minutes postoperatively the patient was noted to be cyanosed and had to be resuscitated. Twelve hours later she collapsed and never recovered, dying 13 hours later. A thorough autopsy revealed multiple fat emboli filling the alveolar capillaries in the lung but no fracture was found. A diagnosis of systemic lupus erythematosus was made on the basis of (i) a history of miscarriage and fulminating pre-eclampsia, (ii) histological renal abnormalities consistent with systemic lupus, (iii) pulmonary fat emboli and (iv) anti-nuclear antibody at a titre of 1:20 in stored blood taken from the patient when she was pregnant. None of these factors is diagnostic of systemic lupus, so the cause of death must remain in doubt. However, the hysterotomy is likely to have been a major factor contributing to death. The decision to perform hysterotomy was unusual since, at the time, prostaglandin termination was well accepted and widely practised.

Blood diseases

i. *Iron deficiency anaemia*

An Asian patient, a multipara, was diagnosed as suffering from iron deficiency anaemia in the second trimester when a haemoglobin concentration of 6.6g/dl was detected. A stool specimen showed hookworm ova and Giardia lamblia cysts. At 27 weeks gestation four units of packed red cells were transfused over 24 hours and on the following day, an iron dextran infusion was commenced. After 2½ hours she complained of weakness and paraesthesia, and after 11 hours developed convulsions and died. A CT scan of the brain performed before death showed diffuse symmetrical cerebral swelling which could have been associated with sagittal sinus thrombosis but this could not be confirmed because permission for autopsy was not given so the precise cause of death is thus unknown but it might have been due to a reaction to the iron dextran infusion.

ii. *Aplastic anaemia*

A young Caucasian woman, who had had idiopathic aplastic anaemia since the age of 9 years and required regular blood transfusion, became pregnant for the first time at the age of 21 years. Transfusions were continued during pregnancy under the close supervision of the haematologists, as well as the consultant obstetric team. Following an episode of haematemesis and melaena, pre-term labour began at 34 weeks and a live infant was spontaneously delivered. Although the mother's condition was initially satisfactory, it deteriorated and she died five days later of a febrile illness, despite all therapy. Autopsy revealed that death was due to *E. coli* septicaemia. This death has also been described in Chapter 7, in which the very high risk of mortality associated with pregnancy in women with aplastic anaemia is stressed.

Diseases of the central nervous system

i. *Intracranial haemorrhage*

There were 11 deaths from intracranial haemorrhage unrelated to hypertensive disease of pregnancy in this triennium. Up to 1976, such deaths were classified as Fortuitous but a review in the 1979–81 report which was extended to earlier trienna revealed that many were associated with rupture of a berry aneurysm at a younger age than would be expected in non-pregnant females. There is increasing evidence that pregnancy predisposes to aneurysm formation in cerebral and splenic arteries and in the aorta (see page 115), and it seems justifiable to continue the practice started in the last report, of classifying these deaths as Indirect rather than Fortuitous.

Three patients died undelivered. Postmortem Caesarean section was performed in one who collapsed at home at 36 weeks and was found to be dead on admission to hospital. One other patient was admitted to hospital when she collapsed at 28 weeks but within five days she was brain dead and post mortem Caesarean was not performed. The third patient died at home during the 8th week of pregnancy. These women were all under 30 years old and in their second pregnancy. Autopsy revealed, respectively, cerebral haemorrhage, intraventricular haemorrhage and cerebellar haemorrhage, the first two associated with berry aneurysms.

In two patients, signs developed during pregnancy or labour. The first was a 16-year-old primigravida who was admitted at the 37th week because of headache, vomiting and convulsions. Eclampsia was suspected and she was delivered of a live infant by elective Caesarean section. There was however, no hypertension, proteinuria or other evidence of hypertensive disease of pregnancy. After delivery she did not recover consciousness and was maintained on a life support system for five days until her death. Lumbar puncture was not performed. An autopsy, which was considered to be substandard because no histological examination was performed, revealed a left occipital lobe haemorrhage without obvious cause. The second patient, a 20-year-old primigravida, first developed signs of intracranial haemorrhage during the first stage of labour at 36 weeks. Although a live infant was delivered by forceps, the mother died 45 minutes later. Autopsy revealed subarachnoid haemorrhage associated with a berry aneurysm.

In six patients, signs first developed after delivery, 2, 8 and 18 hours and 9, 10 and 14 days post-partum. Autopsy performed in all showed that the first, fourth and sixth deaths were due to subarachnoid haemorrhage resulting from rupture of berry aneurysms. In the second death, cerebral haemorrhages in the right parietal and left occipital lobes were not associated with any aneurysm. In the third death, hypertension developed in labour and worsened after delivery. Eighteen hours post-partum, an intracerebral haematoma was evacuated, but a second intracranial haemorrhage followed, causing death. Autopsy revealed a malformation of the middle cerebral artery. In the fifth death the patient had aborted spontaneously at 25 weeks after a third mid-trimester intrauterine death. She was readmitted 10 days later, unconscious, with a history of right-sided facial pain followed by weakness and slurred speech. She died on the following day. Autopsy revealed extensive haemorrhagic infarction of the right cerebral hemisphere associated with a congenital venous cerebrovascular malformation. The 5 patients died 3, 5, 15, 16, 11 and 14 days respectively after delivery.

While the reduction in deaths from intracranial haemorrhage from 25 in 1979–81 to 11 in 1982–84 at first sight suggests therapeutic improvement, a review of the findings in the Confidental Enquiries since 1970 indicates that this is most likely to be due to chance, because of variation between the triennia. In the 5 triennia between 1982–84 and 1970–72 the number of deaths has been 11, 25, 24, 9 and 23 respectively.

ii. *Cerebral thrombosis and infarction*

Cerebral thrombosis can be arterial, following a thrombus in an internal carotid artery or in a vessel arising from the circle of Willis, the basilar or the vertebral arteries, or venous, following thrombosis in the sagittal or other main venous sinuses. Arterial and venous thromboses both lead to infarction of the brain, but the naked eye appearances of the infarcts are different and readily distinguishable at autopsy. Arterial thrombosis occurs in men and in non-pregnant women and its occurrence in pregnancy may be considered as an Indirect or a Fortuitous cause of death according to circumstances. Venous sinus thrombosis which is rare in non-pregnant women in the 15–45 year age-group, apart from recognised nasal sinus infection, is a well recognised complication of the puerperium. For this reason, such deaths are now considered as Direct and have been counted in Chapter 9.

There were five deaths due to cerebral infarction consequent on arterial thrombosis. Four presented with either sudden hemiparesis or 'fits' and one died without warning. All five were delivered preterm; one by spontaneous abortion, one by vaginal delivery, and three by Caesarean section. Three had clinical evidence of infarction before delivery. A cerebrovascular abnormality was present in one patient. One death occurred from a carotid artery thrombosis on the 10th day after Caesarean section for fetal distress in a woman who had a previous history of pulmonary infarction. On review, we consider that this patient, who had had one intrauterine death and three abortions prior to the pregnancy which proved fatal, could have had circulating lupus anticoagulant[1] which is a recognised cause of intravascular coagulation and is strongly associated with recurrent abortions and intrauterine deaths. It is likely that the carotid artery thrombosis and the previous pulmonary infarct were also related to this underlying pathology.

iii. *Epilepsy*

There were seven deaths of women who developed status epilepticus during or immediately after pregnancy. Contrary to previous reports all these cases have been classified as Indirect because of the known increase in frequency of fits in some epileptic women when they become pregnant. Details of these women are given in Table 14.2. All were aged under 35 years.

Table 14.2 *Details of women thought to have died during or after pregnancy as a result of an epileptiform fit*

ICD Code	Age (years)	Duration of epilepsy (years)	Time of death	Presence of convulsions	Cause of death
Indirect					
648.9 345.9	<25	6	17 weeks of pregnancy	+	Drowned in bath
648.9 345.1	>25	2	? 30 weeks of pregnancy	+	Acute asphyxia
648.9 345.1	>25	?	31 weeks of pregnancy	+	Inhalation of vomit
648.9 345.3	<25	?	3 days postpartum	?	Drowned in bath
648.9 345.1	<25	5	15 days postpartum	+	Acute asphyxia
648.9 345.1	<25	15	16 days postpartum	+	Cardio-respiratory failure
648.9 345.1	>25	17	24 days postpartum	+	Cardio-respiratory failure

All seven women, classified as Indirect deaths, were relatively young (range 18–32 years) and multiparous. Death occurred during pregnancy in three and within four weeks of delivery in four. In all of them, death was due to the convulsions leading to acute asphyxia. All were known to be epileptic when they became pregnant and were taking anticonvulsant drugs throughout pregnancy. Blood levels of anticonvulsant drugs were monitored before death in only one patient and in that case the fact that the serum phenytoin concentration was well below the therapeutic range was disregarded. In a further two cases, the concentration of anticonvulsant drugs in blood taken at autopsy was also below the therapeutic range.

The histories of these deaths suggest that most obstetricans and physicians are still content to allow pregnant epileptics to continue on the same anticonvulsant medication prescribed before pregnancy despite the known increased frequency of convulsions in pregnancy ([2,3]). Progressive haemodilution, the increased frequency of vomiting, and the altered liver metabolism, all tend to lower the blood concentration of orally ingested anticonvulsant drugs. Lack of sleep is thought to be a major factor responsible for inducing a convulsion. The fact that a woman has not had a fit for over a year is no safeguard that she will not have one once pregnant. This is exemplified by the two women in this triennium who died from convulsions, neither of them had had a fit for over a year.

The relatively unchanged number of deaths amongst pregnant women who have died from epilepsy since 1970 confirm that epilepsy is a dangerous condition. Women with this condition require the advice of a neurologist, pre-pregnancy counselling, and careful planning and management of therapy during pregnancy. It is not always appreciated that routine folic acid and iron medication antenatally can counteract the therapeutic effect of anticonvulsants, which are antifolates. Drug levels should be monitored monthly as it is likely that dosage will need to be increased. In labour, anticonvulsant therapy must be continued. Blood levels should be checked in the first 24 hours after delivery and the dose reduced according to blood levels over the puerperium. All pregnant women with epilepsy should be advised that taking a bath without informing someone and keeping the door unlocked is potentially dangerous, because of the risk of convulsions when unattended.

Diseases of the circulatory system

i. *Aneurysms*

Bleeding from an arterial aneurysm in women who have not been suffering from hypertensive diseases of pregnancy has accounted for 67 deaths between 1970 and 1984 (Table 14.3). The three most common sites in order of frequency are cerebral arteries (34 cases), ascending aorta (19 cases) and splenic artery (10 cases). The total of 34 ruptured berry aneurysms is probably an underestimate since in some women with intracranial haemorrhage no autopsy was performed and in others the report was inadequate. Seventeen of the 19 aortic aneurysms had dissected and ruptured. Five were positively associated with Marfan's syndrome; in four the association had been excluded and in 10 the reports available were inconclusive. In one only was Ehlers-Danlos syndrome (type IV) suggested though the appearances described in some of the others was consistent with this condition. Fifteen of the aneurysms were known to involve the asceding aorta; one was known to be abdominal and the site of three was not described. The association of ascending aortic involvement with dissection and rupture in an age group under 40 years strongly suggests medionecrosis as a cause. The total of 10 splenic aneurysms is remarkable as these are virtually unknown outside pregnancy.

Table 14.3 *Aneurysms in the triennia, 1970–1984*

Site	Triennium					
	70–72	73–75	76–78	79–81	82–84	Total
Aorta with Marfan	1	2	–	1	1	5
Aorta without Marfan	–	–	2	–	2	4
Aorta, Marfan not specified	5	3	–	2	–	10
Splenic	6	1	1	2	–	10
Cerebral	10	2	8	8	6	34
Coronary	–	–	–	1	1	2
Other (liver, renal)	–	–	2	–	–	2
Total	22	8	13	14	10	67

These findings indicate a relationship between pregnancy and degenerative diseases of certain arteries. It is well recognised that in the so-called berry aneurysms there is a congenital weakness in the musculo-elastic arterial walls which, particularly in the presence of hypertension, can lead to aneurysm formation and rupture in later life, in both sexes. Aneurysms involving the ascending aorta also occur, less commonly and at a somewhat later age, in men and non-pregnant women, usually associated with defects in the musculo-elastic connective tissues of the arterial wall. Two explanations for the greater prevalence and earlier presentation in pregnancy seem possible. There might be a specific connective tissue disorder associated with pregnancy which produces arterial weakness. Alternatively, there can be intra-arterial pressure variations associated with pregnancy, and labour might cause aneurysm formation in an area of weakness which already exists. Further research is needed on arterial changes in pregnancy.

In this triennium, the three women who died from ruptured aortic aneurysm are described below. Six women died from rupture of a cerebral berry aneurysm and are described in the section on intracranial haemorrhage. There were no deaths from ruptured splenic aneurysm.

ii. *Dissecting aortic aneurysm*

Three deaths were due to rupture of a dissecting aortic aneurysm.

One patient, para 5, collapsed and died unexpectedly, having been admitted apparently in labour when 38 weeks pregnant. Autopsy revealed a ruptured dissecting aneurysm and other stigmata of Marfan's syndrome. This is a disorder of connective tissue associated with tall stature, muscular hypotonia, a high arched palate, short sightedness with subluxation of the lens, arachnodactyly, and dilatation of the aorta which can be followed by medionecrosis and dissection of the aorta to form an aneurysm; the condition is inherited as an autosomal dominant.

A second patient, a para 3, was admitted to hospital when 35 weeks pregnant with unexplained epigastric pain. She was allowed home a week later when the pain appeared to have resolved, but was readmitted moribund on the same day.

A third patient, also a para 3 collapsed and died after spontaneous delivery of a live infant at term.

In these last two women a ruptured dissecting aortic aneurysm was found at autopsy, but in neither was there any evidence of Marfan's syndrome. It is possible that they had Ehlers-Danlos syndrome, type IV, which can also be associated with dissecting aneurysm, but there is no suitable confirmatory histological report available.

Diseases of the respiratory system

i. *Pneumonia*

Two maternal deaths were due to pneumonia.

The first was a multipara, who was first seen at about 30 weeks of pregnancy by her local general practitioner, having had no previous antenatal care. She was sent to hospital with a painless antepartum haemorrhage. She also had an upper respiratory tract infection for which she was treated with ampicillin. The following day she went into premature labour and had an uncomplicated delivery. Eight hours after delivery she was diagnosed as having right lobar pneumonia and was transferred to a medical ward. Twenty-one hours after delivery she became cyanosed and, despite all efforts to resuscitate her, she died. Autopsy confirmed the clinical diagnosis. The failure to respond to treatment and the rapid deterioration after delivery were ascribed to her poor social circumstances and physical condition. She was also a heavy smoker.

The second death was of a young woman who was admitted to hospital in early pregnancy with a one week history of a sore throat, a productive cough and vomiting. Two days later she collapsed and a diagnosis of right lobar pneumonia was made. The following day she was found dead in bed. Autopsy reported an acute suppurative pneumonia attributed to a virus infection.

Sudden unnatural deaths

These included nine coroners' cases in which the verdicts were suicide in five, accidental death in one, misadventure in two and an open verdict on one.

These terms which are among those the coroner may use to describe a maternal death, merit a short comment.

The verdict 'suicide' or 'she took her own life' is generally used only when the evidence of intent is incontrovertible. 'Accidental death' is used when death is due to unforeseen extraneous circumstances, while 'death by misadventure' indicates that, while death was accidental, the deceased or another might have contributed to it. The terms are used synonymously for statistical purposes and many coroners use them as alternates without distinction. An 'open' verdict is given either when the circumstances under which the patient died cannot be determined to the coroner's satisfaction or when the pathologist cannot establish a cause of death.

i *Suicide*

There were 5 cases of suicide, 2 antenatal and 3 postnatal.

A primigravida with a long history of psychiatric difficulty was initially distressed to have become pregnant but later appeared to accept the situation. She was found dead from carbon monoxide poisoning and ingestion of diazepam. The second case was of a schoolgirl who appeared to have accepted her pregnancy and made arrangements for the baby to be adopted. After an argument at 28 to 30 weeks of pregnancy she unexpectedly committed suicide.

The three postnatal deaths included a young primigravida who concealed her pregnancy until near term. When she informed her family the reaction was hostile. She took an overdose of paracetamol and never recovered consciousness. A stillborn infant was delivered, and she died the following day. Another young primigravida was noted to be exceptionally withdrawn following a pre-term delivery. Psychiatric assessment suggested that long-term supervision was required, but that the need was not urgent. She was allowed home and committed suicide four days later by lying in front of a train. The third patient with a long history of manic-depressive psychosis killed both herself and her baby 25 days after delivery by drowning in a bath.

ii *Accidental death*

A patient required Caesarean section for disproportion because the infant was very large. Blood loss was greater than average and two units of blood were transfused. Severe disseminated intravascular coagulation developed with secondary postpartum haemorrhage, intra and retroperitoneal haemorrhage. Despite transfusion of further large amounts of blood, fresh frozen plasma and laparotomy she died two hours later. Death was due to the first unit of blood having been infected with *pseudomonas fluorescens* which can multiply at 4°C. Since it is the toxin produced by the organism which causes the coagulation failure, antibiotics are unlikely to retrieve the situation once the blood has been transfused. There is no realistic means of routine screening each unit of blood given before transfusion. Whenever a unit of blood has been transfused a remnant should be kept for 24–48 hours in case subsequent investigation is necessary.

iii. *Misadventure*

In one patient laparoscopic sterilisation was planned following vaginal suction termination of pregnancy in a grand multipara. When carbon dioxide insufflation was commenced after suprapubic insertion of the Verres' needle, gas was noted to be passing from the cervix. Although the needle was withdrawn and resited, the patient was soon found to be cyanosed and pulseless. Death was attributed to gas embolism but since autopsy was not performed until 4 days after the death and was substandard this diagnosis could not be confirmed. Attention has also been drawn elsewhere to the dangers of concurrent sterilisation and pregnancy termination (Chapter 11), but inserting the Verres' needle suprapubically to administer the carbon dioxide should be avoided because of the danger of the tip entering a large, vascular and recently evacuated or puerperal uterus.

The second patient died 27 days after the birth of her third infant. A history of severe depression after a previous delivery had not been drawn to the attention of the obstetrician. Death was due to hepato-renal failure from a self-adminstered drug overdose.

iv. *Open verdict*

In one case the coroner gave an open verdict. A primigravida developed acute depressive puerperal psychosis a few days after delivery of a live infant at term and was seen by the psychiatrist. When he left the room to get suitable medication, she jumped from the second floor window and died from her injuries.

References

1 Lubbe N F, Butler W A, Palmer S J, Liggins G C. Lupus anticoagulant in pregnancy. *Brit J Obst and Gynae* 1984; **91**: 357–63.

2 Hopkins A. Epilepsy and anticonvulsant drugs. *Brit med J* 1987; **294**: 497.

3 Knight A H, Rhind E G. Epilepsy and pregnancy. *Epilepsia* 1975; **16**: 99.

15. Fortuitous deaths

Fortuitous deaths are deaths from non-obstetric causes which happen to occur in pregnancy or the puerperium but are not considered to be related to the pregnancy. They are excluded from maternal mortality as internationally defined. These deaths are listed in Table 15.1.

In 1982–84 there were 34 fortuitous deaths. These included three cardiac deaths which were also considered to be Fortuitous and are mentioned in Chapter 10.

Table 15.1 *Fortuitous deaths*

Number	Cause of death
Infectious disease	
1	Miliary tuberculosis
1	Varicella pneumonitis
Neoplastic disease	
1	Carcinoma of stomach
1	Carcinoma of liver
1	Glucagonoma of pancreas
2	Carcinoma of bronchus
2	Carcinoma of kidney
1	Cerebral glioma
1	Cerebellar ependymoblastoma
1	Cerebellar medulloblastoma
1	Cerebellar astrocytoma
1	Cerebral astrocytoma
1	Nodular goitre of thyroid causing tracheal obstruction
Blood disease	
1	Aplastic anaemia
1	Acute thrombocytopenic purpura
Disease of nervous system	
2	Acute meningitis secondary to otitis media
1	Epilepsy
Disease of Circulatory System	
2	Myocardial infarction
1	Mesothelioma of atrio-ventricular node
Disease of respiratory system	
1	Viral pneumonia
1	Bronchopneumonia

Table 15.1—*continued*

Number	Cause of death
Sudden unnatural deaths	
Accident	
3	RTA Multiple injuries
1	Asphyxia due to inhalation of vomit
Suicide	
1	Overdose
Manslaughter	
2	Multiple wounds to head and neck
Murder	
1	Multiple head wounds
1	Stab wounds in chest
Total 34	

16. 'Late' deaths

Deaths in women more than 42 days, but less than one year, after pregnancy or delivery, are defined as Late deaths. In 1982–84 there were 73 Late deaths. These deaths are excluded under the International Definition of Maternal Deaths. Six of the deaths were considered to be Direct, 17 to be Indirect and 50 to be Fortuitous. This compares with a total of 66 Late deaths in 1979–81 of which 5 were Direct, 13 Indirect and 48 Fortuitous.

Direct maternal deaths

Six of the Late deaths were considered to be Direct.

Three of the deaths were due to pulmonary embolism. The first woman was aged 24 years and para 2. She was admitted to hospital at 14 weeks gestation by dates, for evacuation of retained products of conception after an ultrasound scan had confirmed a missed abortion. Five weeks later she developed chest pain and a cough. A chest x-ray was reported as showing inflammatory changes at both bases. Eleven days later, despite treatment with antibiotics by her general practitioner, she developed acute dyspnoea and died one hour after hospital admission. Autopsy revealed a pulmonary embolus arising from a deep pelvic vein thrombosis. There were also two infarcts in the lower lobes of the lungs apparently a few days old.

The second woman, aged 38 years and para 4, had a normal delivery at term. When she was seen by her general practitioner six weeks after delivery she was prescribed antibiotic treatment for a chest infection. She was admitted to hospital 3 weeks later in renal failure with increasing dyspnoea and oedema. She died within a few hours of admission, 63 days after delivery. At autopsy there was evidence of massive pulmonary infarction with related obstructive pulmonary hypertension and widespread venous thrombosis including the renal veins.

The third woman, a primigravida aged 32 years, had a normal delivery at 38 weeks gestation. Eight weeks after delivery she developed dyspnoea and wheezing, which was treated at home until she had a haemoptysis after which she was admitted to hospital. There was evidence of venous thrombosis and pulmonary embolism which was treated with anticoagulants. She died three weeks later after slow deterioration in her condition and with evidence of right heart failure.

Cardiomyopathy was suspected at autopsy but not confirmed on histological examination, and death was considered to be due to recurrent pulmonary emboli.

One Late death was ascribed to complications of pre-eclampsia. The patient, a primigravida aged 27 years, had an elective Caesarean section at 30 weeks gestation for fulminating pre-eclampsia. Pre-operatively there was evidence of pulmonary oedema, so post-operatively, she was maintained on ventilation with a tracheotomy. A suspected respiratory infection was treated with antibiotics. After two weeks the antibiotics were replaced by steroids, and her respiratory condition improved. However, when sedation was reduced she did not recover consciousness. Neurological diagnosis of basilar artery thrombosis was confirmed by ultrasound scan. She died 70 days post-partum. No autopsy was performed.

One woman died 58 days after a normal delivery, from hypertrophic cardiomyopathy. She was aged 28 years and para 2. Her condition had been diagnosed four years earlier before her first child was born. She was seen by a cardiologist at 26 weeks gestation and her cardiac condition was considered to be satisfactory. Autopsy with histological examination of the heart confirmed the original diagnosis. This case is mentioned in Chapter 10.

The sixth patient died 98 days after an elective Caesarean section for hypertension at 38 weeks gestation. She developed aspiration pneumonia as a result of complications of the anaesthetic, and her case is discussed in Chapter 13.

Indirect and Fortuitous late deaths

There were 17 Indirect maternal deaths and 50 Fortuitous deaths. They are listed in Tables 16.1 and 16.2 respectively.

Table 16.1 *Indirect maternal deaths*

Number	Cause of death
Neoplastic disease	
1	Carcinoma breast
3	Carcinoma cervix
Disease of nervous system	
1	Epilepsy
Disease of circulatory system	
2	Rheumatic heart disease
2	Subarachnoid haemorrhage
Disease of respiratory system	
1	Chronic bronchitis
Disease of genitourinary system	
1	Renal failure

Table 16.1—*continued*

Number	Cause of death
Sudden unnatural death	
Suicide*	
1	Overdose
1	Fall under train
2	Hanging
2	Burns
Total 17	

*All with a history of post-natal depression

Table 16.2 *Fortuitous deaths*

Number	Cause of death
Infectious disease	
1	Staphylococcal septicaemia
1	Herpes simplex pneumonia
Neoplastic disease	
1	Carcinoma colon
1	Carcinoma rectum
1	Carcinoma pancreas
1	Melanoma
1	Cerebellar medulloblastoma
1	Cerebral astrocytoma
3	Disseminated carcinomatosis
2	Lymphosarcoma
1	Lymphoma
Blood disease	
1	Myelomonocytic leukaemia
Disease of nervous system	
1	Encephalitis
1	Epilepsy
Disease of circulatory system	
2	Coronary thrombosis
2	Acute myocarditis
4	Subarachnoid haemorrhage
1	Intracerebral haemorrhage
1	Polyarteritis nodosa
Disease of respiratory system	
3	Acute asthma
Disease of digestive system	
1	Diaphragmatic hernia (with obstruction)
Disease of genitourinary system	
1	Calculus of kidney
1	Pelvic infection
Disease of connective tissue	
2	Systemic lupus erythematosus
Congenital anomaly	
1	Congenital cerebral cyst

Table 16.2—*continued*

Number	Cause of death
Sudden unnatural deaths	
Accident	
1	Road traffic accident
1	Aircraft accident
1	Accidental poisoning
2	Accidental carbon monoxide poisoning
1	Accidental drowning
Suicide	
3	Overdose
1	Hanging
1	Fall from building
Manslaughter	
1	Gunshot wounds
Open Verdict	
1	Fall from building
1	Hanging
Total 50	

Table 16.3 *The interval in days between delivery or abortion and death of the late cases, 1982–84*

Days between delivery or abortion and death	Direct deaths	Indirect deaths	Fortuitous deaths	Total
43–91	5	8	15	28
92–182	1	4	21	26
183–273	–	2	7	9
274–365	–	3	7	10
Total	6	17	50	73

Table 16.4 *The interval in days between delivery or abortion and death of the late cases, 1976–84**

Days between delivery or abortion and death	Direct deaths	Indirect deaths	Fortuitous deaths	Total
43–91	11	23	36	70
92–182	7	16	58	81
183–273	2	7	25	34
274–365	3	3	24	30
Total	23	49	143	215

*1976–78 was the first triennium when associated diseases were divided into Indirect and Fortuitous deaths.

Table 16.3 shows the interval in days between delivery or abortion and death for the three groups of late cases in 1982–84. Table 16.4 shows the same information for the three triennia since 1976.

As would be expected, more of the deaths considered to be Direct or Indirect deaths occurred in the first six months after delivery compared with the Fortuitous deaths. There are fewer Fortuitous deaths in the second half of the year after delivery compared with the first six months, when they might be expected to occur evenly throughout the year. This is almost certainly an indication of reduced reporting of these cases to the Enquiry.

17. The maternal autopsy

The quality of the autopsy

In the last report, the chapter on the role of the pathologist emphasised that, following a maternal death, failure to provide the clinician with a report based on an adequate autopsy, with the appropriate investigations including histology, should be regarded as substandard practice. Many maternal autopsies are done for the coroner who requires only a registrable cause of death. However finding the cause of death in these circumstances is often a complex problem because there may be multiple causes and many different factors contributing to the deaths. However the Central Assessors therefore consider that the pathologists have an additional responsibility to their clinical colleagues to provide as much information as possible in cases of maternal death, if the reasons for those deaths are to be fully understood. In the 1979–81 triennial report and in a recent scientific publication suggestions were made as to the particular points to be looked for at autopsy, and the appropriate blocks to take for histology. Subjective assessments on the adequacy or otherwise of particular categories of autopsy report were also tabulated.

It is pleasing to record that, in this triennium, the standard has greatly improved. This is due in no small measure to a growing appreciation of the importance of comprehensive maternal autopsies, better collaboration between regional assessors in obstetrics, anaesthetics and pathology, and to the efforts of the individual regional assessors. Tables 17.1 and 17.2 represent subjective assessments designed to assess, more accurately than in the last triennium, the adequacy of reports on Direct and Indirect maternal deaths.

It is clear that the standard of the macroscopic autopsy has improved; out of a total of 127 autopsies on Direct deaths and 61 on Indirect deaths (excluding late deaths), 105 and 45 respectively were satisfactory and many reached a high standard. The figures of 22 Direct (18 per cent) and 16 Indirect (26 per cent) deaths in which the autopsy was substandard leave room for improvement. The four most common defects were the failure to record organ weights, failure to look for or record the source of pulmonary emboli, failure to look for or record the presence or otherwise of berry aneurysms in cerebral or subarachnoid haemorrhage and failure to describe accurately the cardiac anomalies present in congenital heart defects.

The greatest single example of substandard care however is the failure to provide an adequate histological report. There appear to be two main reasons for this. First, adequate material might not have been taken at autopsy. Second, the material is either not processed, or not examined, or the report does not reach the clinician. Enquiries by the regional assessors indicate that the second is more common that might be expected. Every effort should be made to treat the reporting of histology on maternal deaths as urgent and routinely to send a duplicate set of sections or blocks to the regional assessor. Lack of histological confirmation was particularly noticeable in hypertensive disease of pregnancy (13 out of 24), pulmonary embolism (21 out of 24) and disorders of the central nervous system (16 out of 25); for the first of these it was essential for diagnosis and was desirable in the other two.

Requirements for a satisfactory autopsy

The requirements for a satisfactory autopsy have been described[1] and are reiterated here.

1. The principal organs should be fully and accurately described.

2. The weights of heart, liver, spleen, each kidney and brain should be recorded.

3. An especially detailed examination should be made of:—

    a. The genital tract to include a description of tears, lacerations, haematomas and any evidence of adherent placental remnants.

    b. The oesophagus, trachea, bronchi and lungs of patients in whom death has been attributed to amniotic fluid embolism, pulmonary embolism or anaesthetic complications. When a life support machine has been used, the possibility of a pneumothorax following the insertion of central venous pressure lines must be remembered and looked for as the chest is opened. A careful search must always be made in deaths from pulmonary embolism for the source of the embolus.

4. Tissue blocks should be taken for histology routinely from heart (left and right ventricles), lungs (one from each lobe), liver, both kidneys, placental bed (two blocks), placenta when available and any other sites of obvious disease. Not all blocks taken need necessarily be examined immediately but histological examination sufficient to confirm the provisional anatomical assessment must always be made, eg sections of lung in suspected amniotic fluid embolism and of the placental site, kidneys and liver in all cases of hypertension. These confirmatory histological reports should be sent out as soon as possible and preferably with the anatomical report.

5. Cases of sudden or unexplained death need especial care. Blood should be taken for culture in all cases and for the presence and levels of drugs and toxic substances where indicated. Detailed histological examination of all major organs, including the placenta when available, is also essential.

An autopsy can thus be regarded as completed only when the anatomical description is comprehensive, the necessary organ weights have been recorded, any necessary ancillary investigations are complete, and the histology, where appropriate, is attached to the report and provides sufficient information to confirm the autopsy diagnosis. Macroscopic photography of individual organs or lesions can be of great value for descriptive and record purposes.

Other general recommendations

In general the comments on the pathology investigations necessary in particular diseases made in the triennium 1979–81 remain valid and are re-iterated below. Important additional points which have come to light and which merit mention have been included.

1. Autopsies should be if possible performed on *all* maternal deaths. In four cases in which autopsy was not performed, the cause of death as registered was considered to be incorrect because of lack of pathological evidence. In three others, information which would have been valuable in making or confirming a diagnosis could not be obtained.

2. Before starting the autopsy, the pathologist should make sure that he has an adequate clinical report, particularly in cases performed for the coroner, when information is sometimes less than optimal.

3. When death has occurred before, during or within 24 hours of delivery, every attempt should be made to secure the placenta for examination. A careful autopsy should be done also on any stillborn child and the records made available to the pathologist who performs the maternal autopsy.

4. In all sudden and 'unexplained' deaths, blood should be taken at autopsy for bacterial, viral and where necessary, toxicological studies, including the levels of any drugs used. In at least two deaths an infective element was suspected but could not be proven because the necessary investigations were not done. When blood has been transfused the haematology laboratory should be informed of the death and asked to culture residual blood from any container and to check the cross-match. Full histological examination of all organs is essential and particular attention should be given to the pulmonary vasculature to exclude evidence of hypertension.

5. It is helpful if individual pathologists send spare blocks or sections from any maternal autopsy to the regional assessor in pathology.

Recommendations in particular causes of death

Hypertensive diseases of pregnancy. In all patients who die from or with hypertension, tissue blocks must be taken and examined from both kidneys, uteroplacental site and liver. In particular, fibrinoid necrosis, with lipophages and mononuclear cuffing in uterine vessels, provides valuable evidence of pre-eclampsia. In this triennium, 13 out of 21 autopsy reports did not provide enough histological information for eclampsia or pre-eclampsia to be diagnosed with certainty.

Haemorrhage. In all patients who die of, or whose death is associated with haemorrhage, an assessment of haematological studies made during life, with a careful histological search for the deposition of platelets and altered fibrinogen in arterioles and capillaries, must always be made for evidence of disseminated intravascular coagulation, though these changes are not always recognisable. Two of the eight autopsy reports did not provide this information. If hysterectomy has been done to control bleeding, both lungs and uterine vessels must be examined histologically for evidence of amniotic fluid embolism.

Pulmonary embolism A careful search at autopsy for the site of origin of the embolus is mandatory and must include pelvic, femoral and calf veins; this was not recorded in seven of 21 autopsies. Although histological studies are not mandatory it is wise to retain blocks of lung tissue as questions arise, particularly when lung lesions of different ages are present, as to whether there have been multiple emboli or thromboses within pulmonary veins.

Ectopic pregnancy. In all deaths associated with ectopic pregnancy a careful histological examination of *both* tubes is essential to assess previous inflammation and fibrosis. In tubal pregnancies, the tube in which the ectopic pregnancy lies should be sectioned above and below the pregnancy site to look for evidence of previous tubal disease. A careful search should be made also for the presence of an intra-uterine contraceptive device.

Amniotic fluid embolism. The standard of all of the autopsies on patients in whom this diagnosis was suspected was high and no comment is needed. Squames can often be found in blood smears taken from the pulmonary trunk, but the use of frozen section of lung tissue to detect them is not reliable.

Sepsis. It is important always to take appropriate material for micro-biological investigation even when this has been done during life. Blood for culture is best taken by aspiration with a sterile syringe and needle from the heart at the start of the autopsy. Tissue blocks for histology should be examined from all suspected sites of local infection.

Ruptured uterus, Especial care is needed in describing the precise sites and extent of the tear. Tissue blocks should be taken from the edges to assess its age. If hysterectomy has been performed to control bleeding the pathologist performing the autopsy should make every effort to see or to obtain an accurate report on the resected uterus. In one case the uterus was discarded before such an examination could be made.

Miscellaneous deaths. This group, which includes cases of sudden and unexplained death requires the greatest care. Blood for the various possible investigations should be taken at the start of the autopsy, labelled and put aside; it can be discarded later if not needed. A full quota of tissue blocks must always be taken, even if they are not all required, for subsequent examination.

Cardiovascular disease. In congenital heart disease such as Eisenmenger's syndrome, particular care must be taken in describing the defects present and any surgical repair which may have been attempted. In primary cardiomyopathy of pregnancy a very careful description of the size and shape of the chambers and particularly the outflow tracts is essential. An experienced cardiac pathologist in a personal communication wrote of suspected cardiomyopathies in the 1979–81 triennium. 'Words like dilatation, greatly enlarged etc. are used without any quantitation other than a total heart weight. In some cases this weight is discordant with the description of size and hypertrophy. The blocks of myocardium are random with little attempt at orientation. I would feel that the minimal histology . . . is left ventricle—septum posterior wall, lateral wall, anterior wall; right ventricular free wall, right ventricular outflow, left atrium, right atrium.' Though this may seem a heavy load no one pathologist is likely to see more than one case in a lifetime and the trouble is worth taking.

Aneurysms of aorta, splenic and other arteries occur in pregnancy more frequently than in the non-pregnant female population in the 15–44 years age-group (33 in the period 1970–1984). A careful search must always be made for stigmata of Marfan's syndrome and for evidence of generalised connective tissue disorder and careful histology of the aneurysm is essential.

Abortion. Deaths from abortion can provide a particular problem for the pathologist because there is often no clear cut cause. A detailed search for evidence of sepsis, uterine rupture, coagulopathies, maternal disease and, after the fetal age of 16 weeks, amniotic fluid embolism, are obligatory.

Anaesthesia. The two main causes of maternal death associated with anaesthesia are inhalation of gastric content, and problems with endotracheal intubation. In both, the findings are likely to be complicated by a period of survival on a life-support machine. Careful anatomic study with full histology, and often, appropriate microbiological studies are necessary on oesophagus, trachea, main bronchi and lungs. Even where these are available, it can be difficult or impossible to distinguish between inflammatory changes associated with inhaled gastric contents and those which often follow the use of life support machines.

The Liver in pregnancy. A study of autopsy reports reveals that changes which normally occur in the later stages of pregnancy are not well recognised. Although in deaths in which the liver is not considered to be significantly involved (eg amniotic fluid embolism) its weight and histology are often not recorded. Data collected over the last 3 triennia suggest that the normal liver at term rarely weighs less than 2000 g and can weigh as much as 2500 g. This increase can in part be due to an increased vascularity and blood flow, but varying degrees of fatty change are regularly described: necrosis, in the absence of predisposing conditions, is not a usual feature. It is possible that these changes represent the normal end of a spectrum at the other end of which lies the acute fatty liver of pregnancy; along with the unexpected frequency of arterial aneurysms in unusual sites, it provides an interesting subject for research.

Conclusions

The overall standards of autopsy performance and reporting and of anatomical diagnosis show a considerable improvement over those of the previous triennium. The care and thoroughness with which many pathologists now carry out autopsies with very detailed histology, in order to determine exactly what happened to cause death, is noteworthy. Fuller investigations are still needed in some cases of unexplained death and studies of drug levels would have been helpful on some. Too many reports, however, continue to be substandard either because of lack of anatomical detail, or because of the absence of adequate supportive histology attached to the anatomical report. This is particularly noticeable in patients with hypertensive disease and with suspected disseminated inatravascular coagulation. In some cases, further enquiry by the regional assessor in pathology has produced unreported histological material which would have allowed a more correct final assessment, and it is clear that a good deal more 'hidden' information is available than often appears at first sight. We should like to see implemented the suggestion that duplicate histological material should be sent to the regional assessor in pathology in all maternal deaths.

Reference

1 Rushton D I, Dawson I M P, The maternal autopsy. *J Clin Pathol* 1982; **35:** 909–21.

Table 17.1 *Subjective assessment of the adequacy of autopsies in Direct maternal deaths, 1982–84*

Chapter title	Total deaths	Autopsies			Histology		Other investigations	
		None	Satis	Unsatis	Satis	Unsatis/ no record	Satis	Unsatis/ no record
Hypertensive disease of pregnancy	25	1	20	4	11	13	–	–
Haemorrhage	9	1	7	1	5	3	–	–
Pulmonary embolism	25	4	14	7	2	19	–	–
Ectopic pregnancy	10	1	6	3	4	5	–	–
Amniotic fluid embolism	14	–	14	–	14	–	–	–
Sepsis	2	–	2	–	1	1	–	–
Ruptured uterus	3	1	2	–	–	2	–	–
Miscellaneous	21	1	16	4	12	8	4	9
Abortion	11	2	9	–	6	3	3	–
Anaesthetic deaths	18	–	15	3	8	10	2	–
Totals	138	11	105	22	63	64	9	9

Note: Histology reports listed as unsatis/no record do not include patients on whom an autopsy was not performed. Other investigations have only been assessed when they were considered necessary for adequate diagnosis.

Table 17.2 *Subjective assessment of the adequacy of autopsies in Indirect maternal deaths, 1982–84*

Chapter title	Total deaths	Autopsies			Histology		Other investigations	
		None	Satis	Unsatis	Satis	Unsatis/ no record	Satis	Unsatis/ no record
Septicaemia	3	–	3	–	2	1	2	1
Neoplasms	11	6	5	–	5	–	–	–
Endocrine and immune disorders	4	–	4	–	3	1	–	–
Blood diseases	2	1	1	–	–	1	–	–
Diseases of central nervous system	26	1	18	7	–	16	3	2
Diseases of heart and vessels	14	2	8	4	6	6	–	–
Diseases of respiratory system	2	–	1	1	1	1	–	–
Unnatural death	9	–	5	4	3	7	4	–
Totals	71	10	45	16	20	33	9	3

Note: Histology reports listed as unsatis/no record do not include patients on whom an autopsy was not performed. Other investigations have only been assessed when they were considered necessary for adequate diagnosis.

18. Summary and recommendations

Summary

The number of women who died in the last five triennia relative to the cause of death are shown in Table 18.1. The rates of death for each cause per million pregnancies are shown in Table 18.2.

Once again in 1982–84, pulmonary embolism and hypertensive diseases of pregnancy were the commonest causes of death during pregnancy; anaesthesia and amniotic fluid embolus came third and fourth. However with the exception of pulmonary embolism, all single causes of death have fallen compared with the previous triennia. Over the last five triennia there has been a fairly consistent fall in the rates of death from hypertensive diseases, anaesthesia, abortion, ectopic pregnancy, haemorrhage and sepsis. The most marked falls have occurred in deaths from abortion and sepsis. Deaths from pulmonary embolism have shown less change since 1973–75, and amniotic fluid embolism is relatively unchanged since 1970–72.

The general improvement in maternal mortality which is occurring in all western countries is probably mainly due to the improved quality of care in the maternity services, and the better health of pregnant women throughout the population. Since care was considered substandard in 52 per cent of the Direct deaths in this triennium, it is clear there is still room for improvement.

The continued rise in the number of Caesarean sections is also occurring in other Western countries, but it is a phenomenon which should be kept under review. In this triennium, the actual number of deaths has actually fallen, despite an increase in the number of operations. This excellent outcome is likely to be due to the better quality of care for women undergoing Caesarean section. It should not be forgotten that vaginal delivery is safer than delivery by Caesarean section.

Recommendations

Pulmonary embolism

1. Sudden and unexpected deaths from pulmonary embolus continue to occur and indicate the need for earlier detection of thrombus formation in the veins of the pelvis and legs.

2. There is no evidence in this report that the use of subcutaneous heparin in the treatment of pulmonary embolism has improved the prognosis for affected

Table 18.1 *Causes of Direct maternal deaths/numbers and percentages, 1970–84**

Cause of Direct maternal death	1970–72*		1973–75*		1976–78		1979–81**		1982–84†	
	No	(%)	No	(%)	No	(%)	No	(%)	No	(%)
Pulmonary embolism	51	(14·9)	33	(14·5)	43	(19·8)	23	(12·9)	25	(18·1)
Hypertensive diseases of pregnancy	43	(12·5)	34	(15·0)	29	(13·4)	36	(20·2)	25	(18·1)
Anaesthesia	37	(10·8)	27	(11·9)	27	(12·4)	22	(12·4)	18	(13·0)
Amniotic fluid embolism	14	(4·1)	14	(6·2)	11	(5·1)	18	(10·1)	14	(10·1)
Abortion	73	(21·3)	27	(11·9)	14	(6·5)	14	(7·9)	11	(8·0)
Ectopic pregnancy	34	(9·9)	19	(8·4)	21	(9·7)	20	(11·2)	10	(7·2)
Haemorrhage	30	(8·7)	21	(9·3)	24	(11·1)	14	(7·9)	9	(6·5)
Sepsis, excluding abortion	30	(8·7)	19	(8·4)	15	(6·9)	8	(4·5)	2	(1·4)
Ruptured uterus	11	(3·2)	11	(4·8)	14	(6·5)	4	(2·2)	3	(2·2)
Other Direct causes	20	(5·8)	22	(9·7)	19	(8·8)	19	(10·7)	21	(15·2)
All deaths	343	(100)	227	(100)	217	(100)	178**	(100)	138†	(100)

† Progressive fall in numbers: this triennium is 40% of 1970–72 (PE 49%, HDP 58%, An. 49%, AFE 100%, Ect. 29%, Haem 30%, Sep. 7%, Rupt. 27%).
* For changes in numbers and rates since 1st report see page 153.
**Includes two other Direct deaths omitted in the last report.

Table 18.2 *Direct deaths by cause, rates per million pregnancies, 1970–84**

Cause of direct maternal death	1970–72*	1973–75*	1976–78	1979–81**	1982–84
Pulmonary embolism	17·6	12·8	18·5	9·0	10·0
Hypertensive diseases of pregnancy	14·9	13·2	12·5	14·2	10·0
Anaesthesia	12·8	10·5	11·6	8·7	7·2
Amniotic fluid embolism	4·8	5·4	4·7	7·1	5·6
Abortion	25·3	10·5	6·0	5·5	4·4
Ectopic pregnancy	11·5	7·4	9·0	7·9	4·0
Haemorrhage	10·4	8·1	10·3	5·5	3·6
Sepsis, excluding abortion	10·4	7·4	6·5	3·1	1·0
Ruptured uterus	3·8	4·3	6·0	1·6	1·2
Other Direct causes	6·9	8·5	8·2	7·5	8·4
All deaths	118·7	88·0	93·4	70·0**	55·0

* For changes in rates since last report see page 153.
**Includes two other Direct deaths omitted in the last report.

pregnant women. However, it is recommended that as soon as pulmonary embolism or deep vein thrombosis is strongly suspected, treatment with intravenous heparin should be started.

3. While short term prophylactic treatment with heparin of deep vein thrombosis and pulmonary embolus is recommended, the risks of long term therapy should be kept in mind.

Hypertensive diseases of pregnancy

1. Earlier treatment with hypotensive agents and, if necessary, by early delivery is likely to decrease the risk of death from intracranial haemorrhage.

2. Cerebral complications are the commonest cause of death, indicating that severe hypertension is not controlled effectively. Renal, hepatic or blood coagulation complications also contribute to death. They often arise insidiously and can be more severe, or progress more rapidly than might be expected from the level of hypertension alone, whether or not antihypertensive drugs are used. Repeated measurements of blood urea, creatinine, platelets and liver enzymes are necessary to provide an early indication of the development of these complications.

3. The signs of impending eclampsia are all too often disregarded. The combination of hypertension and significant proteinuria should lead to immediate hospital admission. If there are also symptoms such as headache, visual disturbances, epigastric pain or vomiting the patient should be treated as an acute emergency. When proteinuria is present in a woman with pre-eclampsia, continuation of pregnancy beyond 36 weeks is rarely justified.

4. As cases of life-threatening severity are now rare, most obstetricians and midwives have relatively little experience in their management. The persistence of substandard care reaffirms the necessity for the ready availability of expert advice. It is suggested that advisory sub-committees in obstetrics and gynaecology should draw up plans and make recommendations to their regional health authorities for establishing regional teams.

Anaesthesia

1. The increased number of anaesthetics administered to pregnant women in this triennium means that the true reduction in deaths from anaesthesia is greater than the actual number suggests.

2. There is still a shortfall in the availability of well trained anaesthetists and assistants, facilities and equipment in the maternity services. Until this deficit is made good women will be at unnecessary risk of death from anaesthesia in some units.

3. Earlier consultation and planning between the obstetric and anaesthetic teams is still required, especially when there is a likelihood of some operation or procedure being required for a woman likely to be an anaesthetic risk.

Amniotic fluid embolism

1. There were 14 histologically proven deaths from amniotic embolism in this triennium and one other confirmed case in which death was attributed to ruptured uterus. No woman presented with symptoms before the onset of labour. Analysis of the cases emphasises the increased risks for women in whom labour is induced with prostaglandin pessaries or intravenous *Syntocinon* and particular care is needed when both drugs are administered. When oxytocic drugs are being used the continuous monitoring of uterine contractions is recommended to detect hyperstimulation as early as possible.

2. The treatment of coagulopathy is still controversial. The replacement of blood loss with whole blood or, if this is not available, fresh frozen plasma and packed cells is generally recommended. Administration of heparin or tranexamic acid is potentially hazardous and should be employed only in consultation with a haematologist.

Ectopic pregnancy

1. Despite the increased incidence of ectopic pregnancy, the death rate has fallen. This is likely to be due to improved diagnosis by laparoscopy in combination with ultrasound examination and beta sub-unit hCG assay.

2. All doctors providing primary consultation, including GPs, casualty or admitting officers, or midwifery and nursing colleagues who may be the woman's first contact, need to be aware of the possibility of ectopic pregnancy in a woman complaining of lower abdominal pain of recent onset. In particular, it is important to remember this in any woman of reproductive age with a longstanding infertility, a history of pelvic inflammatory disease, or any form of tubal surgery.

3. In shocked patients immediate laparotomy with control of haemorrhage and rapid blood transfusion are the main life saving measures. In such cases, prior laparoscopy for diagnostic purposes is rarely justified.

4. Bleeding from cervical and cornual pregnancy are often not considered in the diagnosis because of their relative rarity.

Abortion

1. There were 11 Direct deaths from abortion: 4 due to spontaneous abortion, 7 to legal abortion. In addition, 4 Indirect deaths occurred amongst women undergoing legal abortion. There were no recorded cases of deaths from criminal abortion.

2. It is recommended that:—

—blood must be readily available in NHS and private hospitals when legal abortion is done.

—suspected damage to the genital tract during the operation necessitates close observation of the patient for at least 24 hours post-operatively.

—concurrent sterilisation with legal abortion or after evacuation of retained products of conception is a potentially dangerous procedure. If concurrent sterilisation is considered essential it should be done by a senior obstetrician and a senior anaesthetist.

Haemorrhage

1. The number of deaths from haemorrhage was the lowest recorded. Several of the deaths were associated with unusual clinical problems. Substandard care was usually the result of delay rather than specific errors or omissions.

2. Attention is drawn to the potential danger of the low lying anterior placenta particularly in women previously delivered by lower segment Caesarean section. In such women, in whom elective Caesarean section is planned, the placental site should be identifed by ultrasound before operation. If the placenta is anterior and praevia, the risk that it is accreta is high enough to merit special preparations before the operation. An experienced surgeon should perform the operation, the anaesthetist and theatre staff should be alerted to the problem, and crossmatched blood should be available in theatre.

3. An agreed procedure for management of catastrophic haemorrhage with which the medical, midwifery and nursing staff are fully conversant should be available in all maternity units.

Genital sepsis

1. Genital sepsis, at the present time, is no longer a major cause of maternal death and there were only two deaths directly attributable to sepsis.

2. Overwhelming septicaemia may be present in any pregnant woman who collapses unexpectedly. The possibility that a woman who fails to respond to routine antibiotic treatment may have a virulent resistant organism must always be borne in mind.

Uterine rupture

1. Death from ruptured uterus continues to fall, there being only three cases this triennium.

2. Uterine activity should always be monitored when oxytocic drugs are used to induce or accelerate labour so that hyperstimulation can be detected as soon as it occurs and appropriate action taken (see Chapter 6).

Miscellaneous deaths

1. There were 21 of these deaths, and in eight cases no cause for the sudden and unexpected death could be established, despite the high standard of autopsies. The remainder of the deaths were largely due to dangerous and rare conditions which provided clear explanations for death.

Caesarean section

1. There was a further fall in the number and proportion of deaths from Caesarean section despite a rise in the number of women delivered by the operation (10.1 per cent from 8.9 per cent in the last triennium). Overall, the estimated fatality rate for all Caesarean sections was 0.37 per 1000. The risk of death from emergency Caesarean section was more than four times that of the elective operation.

2. The level of communication between junior and senior staff could be improved. Junior staff continue to carry too much responsibility in the management of women undergoing Caesarean section and consultant obstetricians and anaesthetists need to be involved earlier than is often the case.

3. Early involvement of the duty anaesthetist in any case when operative delivery is more than just a possibility, is essential, particularly in obese and short women and in those with a history of difficult intubation. (see Chapter 13)

Indirect deaths

1. The number of deaths from cardiac disease was similar to the previous two triennia.

2. It is recommended that, in epileptic women, blood levels of anticonvulsant drugs should be monitored throughout pregnancy, labour and the early part of the puerperium because of the higher dosages of these drugs required during pregnancy.

3. The additional risks of pregnancy in women with congenital aneurysms is noted.

Late deaths

Seventy-three deaths occurred more than 42 days but less than one year after the end of pregnancy. Six were Direct deaths. Three were a consequence of pulmonary embolus; one was due to complications of pre-eclampsia; one, to cardiomyopathy, and one, to aspiration pneumonia as a result of anaesthetic complications.

Pathology

There was a continued improvement in the quality of autopsies carried out during the triennium. Specific recommendations on the execution of autopsies on women dying during or after pregnancy have been made in Chapter 17.

19. Overview

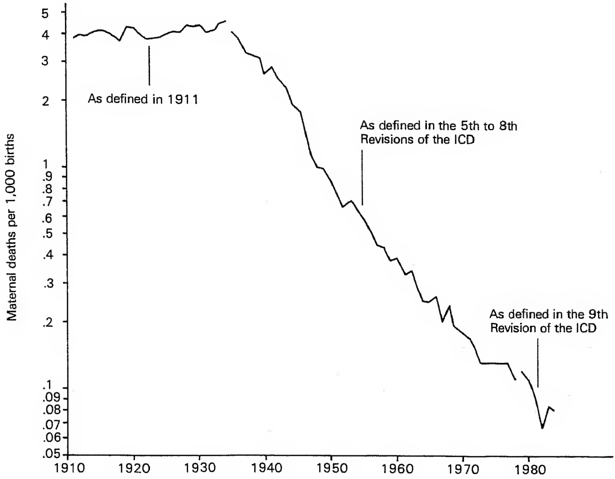
In this, the last Report on Confidential Enquiries into Maternal Deaths for England and Wales (CEMD) it seems appropriate to review the trends in the maternal mortality rate (MMR) between 1952–54 and 1982–84 and the changes in the contribution of various individual causes of death to MMR. Inevitably, much detail must be omitted because there is too much information in the eleven triennial reports to be included in an overview of reasonable length.

Overall maternal mortality rates

Tables 1.3, both for this report for 1982–84 and for 1979–81, show that the MMR has approximately halved every ten years over the period of the enquiries, from 989 per million maternities in 1951 to 86 per million in 1982–84. Figure 19.1 shows that if these rates are expressed on a logarithmic scale, the reduction in MMR over this period was almost linear, indicating that over these years the *rate* of fall in MMR was constant except in the late 1970s. Of course nowadays, the size of the problem, ie number of deaths, is much smaller than it was 30 years ago when there were so many potentially avoidable deaths, but the *rate* of fall in MMR has been maintained. In recent years the numbers of maternal deaths have been so low that apparently large year to year fluctuations in the rate are no greater than would be expected by chance.

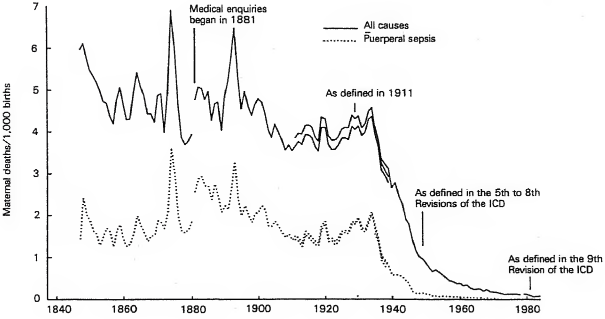
If a longer view is taken of maternal mortality over the last century, the dramatic change in the 1930s becomes apparent. Figure 19.2 illustrates the trends in MMR for England and Wales between 1847 and 1985, showing the sudden reduction which began in 1937, when the MMR was around 4,000 per million. MMR remained on a plateau between 1847 and 1937, despite the introduction of many measures which should have reduced it[1]. These included the techniques of antisepsis and asepsis developed in the 1870s and 1880s; Caesarean section as a safe technique for certain obstetric emergencies after 1900 and especially after 1920; the Midwives' Act of 1902; improvements in the teaching of obstetrics and growing recognition and provision of antenatal care in the 1920s and 1930s. Two other important papers, [2,3] provided convincing evidence that the common major contributions to this persisting high MMR in England and Wales, and elsewhere, in a large proportion of the confinements conducted by general practitioners, were neglect of asepsis and excessive use of forceps under chloroform anaesthesia, the rate of forceps delivery being as high as 70 per cent among some doctors and the procedure not infrequently performed before the cervix was fully dilated. The Registrar-General's Decennial Supplement for 1930–32 showed higher maternal mortality rates among wives of men in professional and managerial occupations than among the wives of manual workers, whose

Figure 19.1 *Maternal mortality, England and Wales 1911–84 plotted on a logarithmic scale.*



Source: OPCS mortality statistics

Figure 19.2 *Maternal mortality, England and Wales, 1847–1984*



Source: OPCS Mortality statistics

deliveries were much more likely to be supervised by midwives than by doctors.

The sharp fall in 1937 was probably initiated by the introduction of sulphonamides with a possible simultaneous reduction in the virulence of the *haemolytic streptococcus* reducing sepsis deaths as illustrated in Figure 19.2[2]. Other factors doubtless made important contributions after the first few years, including safe blood transfusion, ergometrine, penicillin and the new antibiotics, increasing hospital confinement with better training of midwives and steadily increasing professional expertise amongst obstetricians resulting from the foundation of the British (now Royal) College of Obstetricians and Gynaecologists in 1929. Improving maternal health must have contributed also as the country recovered from the depression of the early 1930s, but MMR seems to be more sensitive to the quality of obstetric care in its widest sense than to the socioeconomic circumstances of mothers[1,2,3].

Figure 19.2 shows that by the time CEMD were initiated in 1952, MMR had already fallen to less than one quarter of the 1937 level. The fall in MMR between 1952 and 1984 was relatively insignificant compared with the massive reduction previously achieved. However, the scale of this graph is influenced by the very large numbers of deaths in the earlier years, many of which were potentially avoidable by relatively minor improvements in care. To avoid this, the changes in MMR with time have been expressed on a logarithmic scale in Figure 19.1. This illustrates the rate of change in MMR since 1911. It shows that, ever since MMR began to fall in 1937, the rate of reductions has remained constant, approximately halving every decade. Among the many factors which must have contributed to this constant rate of fall in MMR in England and Wales over the 50 years from 1937, the quality of obstetric care provided by the maternity services must have improved steadily, judging by the sensitivity of MMR to obstetric care. The close association between MMR and the quality of obstetric care underlines the importance of the CEMD in obstetric audit. Despite the steady fall in MMR, however, there can be no complacency. The incidence of substandard care in the 1982–84 report shows clearly that further improvement can be achieved. Continuing careful assessment is required to identify the causes of maternal mortality most likely to be prevented or reduced by better mangement.

Trends in MMR from individual causes of death

Table 19.1 lists the total number of maternities and pregnancies from which the various rates are calculated. In the six triennia between 1952–54 and 1967–69, mortality rates were calculated with 'maternities' as a denominator. 'Maternities' is the number of women who gave birth during the triennia; it does not include the additional numbers due to multiple births. If the latter are counted the total denominator is total births. 'Maternities' is not an appropriate denominator for deaths which occur before fetal viability, such as abortion or ectopic pregnancy. From 1970, the denominator used has been total pregnancies, which are shown in Table 19.1 for the five triennia between

1970–72 and 1982–84. Total pregnancies include ectopic pregnancies and spontaneous and legal abortions, in addition to maternities.

Table 19.2 shows the numbers of death from individual causes by triennia from 1952–54 to 1967–69, and Table 19.3 the numbers from 1970–72 to 1982–84. Table 19.4 shows the Direct maternal death rates per million maternities by triennia between 1952–54 and 1967–79. Table 19.5 shows the Direct maternal death rate per million total pregnancies by triennia between 1970–72 and 1982–84. Despite the change to the more comprehensive denominator in 1970, the *rates* in Tables 19.4 and 19.5 more accurately represent the trends in MMR than do the *numbers* of deaths in Tables 19.2 and 19.3, because of the marked fluctuations in the number of maternities and of total pregnancies over the triennia shown in Table 19.1.

Table 19.1 *Total maternities England & Wales 1952–1984 by triennium.*
**Total pregnancies England & Wales 1970–1984 by triennium*

Triennium	Total maternities	Total pregnancies
1952–54	2,052,953	NA
1955–57	2,113,471	NA
1958–60	2,294,414	NA
1961–63	2,520,420	NA
1964–66	2,600,367	NA
1967–69	2,457,443	NA
1970–72	2,298,201	2,890,768
1973–75	1,921,568	2,578,386
1976–78	1,748,851	2,323,003
1979–81	1,923,735	2,543,233
1982–84	1,883,753	2,507,038

*Includes ectopic pregnancies and spontaneous and legal abortions as well as total maternities.

Table 19.2 *Numbers of Direct maternal deaths by cause 1952–54 to 1967–69*

Causes	1952–54	1955–57	1958–60	1961–63	1964–66	1967–69
Hypertensive diseases of pregnancy	246	171	118	104	67	53
Pulmonary embolism	138	157	132	129	91	75
Abortion	153	141	135	139	133	117
Haemorrhage	188	138	130	92	68	41
Anaesthesia	not classified separately				50	50
					Calculated later, not included in total	
Ectopic pregnancy	no separate chapter			42	42	32
Amniotic fluid embolism	not recognised			27	30	27
Sepsis—excluding abortion	included in abortion			33	57	26
Ruptured uterus	no separate chapter			38	30	18
Other Direct	no separate chapter			88	61	66
	All other 369	All other 254	All other 227			
Total	1,094	861	742	692	579	455

Table 19.3 *Numbers* of Direct maternal deaths by cause, 1970–72 to 1982–84*

	1970–72**	1973–75**	1976–78	1979–81***	1982–84
Hypertensive diseases of pregnancy	43	34	29	36	25
Pulmonary embolism	51	33	43	23	25
Abortion	73	27	14	14	11
Haemorrhage	30	21	24	14	9
Anaesthesia	37	27	27	22	18
Ectopic pregnancy	34	19	21	20	10
Amniotic fluid embolism†	14	14	11	18	14
Sepsis—excluding abortion	30	19	15	8	2
Ruptured uterus	11	11	14	4	3
Other Direct	20	22	19	19	21
Total	343	227	217	178***	138

* late deaths excluded.
** For changes in numbers since last report see page 153.
*** Includes two other Direct deaths omitted in the last report.
† histologically confirmed.

Table 19.4 *Rate of Direct maternal deaths by cause per million maternities, 1952–54 to 1967–69*

Causes	1952–54	1955–57	1958–60	1961–63	1964–66	1967–69
Hypertensive diseases of pregnancy	119·8	80·9	51·4	41·3	25·8	21·6
Pulmonary embolism	67·2	74·3	57·5	51·2	35·0	30·5
Abortion	74·5	66·7	58·8	55·1	51·1	47·6
Haemorrhage	91·6	65·3	56·7	36·5	26·2	16·7
Anaesthesia	no separate chapter				19·2	20·3
					Calculated later, not included in total	
Ectopic pregnancy	no separate chapter			16·7	16·2	13·0
Amniotic fluid embolism	not recognised			10·7	11·5	11·0
Sepsis—excluding abortion	included in abortion			13·1	21·9	10·6
Ruptured uterus	no separate chapter			15·1	11·5	7·3
Other Direct	All other causes			34·9	23·5	26·9
	179·7	120·2	98·9			
Total	532·8	407·4	323·4	274·6	222·7	185·2

1952–54 to 1967–69

Tables 19.1, 19.2 and 19.4 show that the first six triennia, 1952–54 to 1967–79, were dominated by deaths from hypertensive diseases of pregnancy, pulmonary embolism, haemorrhage and abortion. Deaths from abortion, often associated with criminal interference, showed little real improvement over these triennia, and abortion was the main cause of death between 1958–60 and 1967–79. Throughout these triennia, the birth rate rose steadily (Table 19.1) and there was concern about the lack of adequate hospital

Table 19.5 *Rates* of Direct maternal deaths by cause, per million total pregnancies, 1970–72 to 1982–84*

	1970–72**	1973–75**	1976–78	1979–81***	1982–84
Hypertensive diseases of pregnancy	14·9	13·2	12·5	14·2	10·0
Pulmonary embolism	17·6	12·8	18·5	9·0	10·0
Abortion	25·3	10·5	6·0	5·5	4·4
Haemorrhage	10·4	8·1	10·3	5·5	3·6
Anaesthesia	12·8	10·5	11·6	8·7	7·2
Ectopic pregnancy	11·8	7·4	9·0	7·9	4·0
Amniotic fluid embolism†	4·8	5·4	4·7	7·1	5·6
Sepsis—excluding abortion	10·4	7·4	6·5	3·1	0·8
Ruptured uterus	3·8	4·3	6·0	1·6	1·2
Other Direct	6·9	8·5	8·2	7·5	8·4
Total	118·7	88·0	93·4	70·0***	55·0

* Per million total pregnancies (including spontaneous and legal abortion and ectopic pregnancies).
** For changes in rates since last report see page 153.
*** Includes two other Direct deaths omitted in last report.
† histologically confirmed.

facilities for obstetric care. There were often comments in these reports of the dangers of home confinement in women with post partum haemorrhage or other complications; of inadequate anticipation of treatment of severe hypertensive diseases of pregnancy; of the need for better booking, assessment, and arrangements for confinement; and of the need for better flying squad services.

During the first three triennia, 1952–60, only the four main causes of death were described in separate chapters, and all other direct deaths were grouped together. Table 19.4 shows the MMR per million maternities from hypertensive diseases in these triennia were 119.8, 80.9 and 50.4, respectively and from haemorrhage 91.6, 65.3 and 56.7, respectively. The comparable MMR from pulmonary embolism were 67.2, 74.3 and 57.5, and from abortion, 74.5, 66.7 and 58.8. The marked reduction in deaths from hypertensive diseases and haemorrhage might have occurred simply because it was easy to improve on poor previous standards of care. Any such improvements clearly had little effect on deaths from pulmonary embolism or abortion.

It was only in 1961–63 that amniotic fluid embolism was recognised and a separate chapter introduced. In this triennium, separate chapters were also introduced for ruptured uterus, ectopic pregnancy and 'miscellaneous', Direct deaths. In this triennium also, deaths from septic abortion were for the first time included in the Abortion Chapter, having previously been included under sepsis. Between 1952–54 and 1967–69, anaesthetic deaths did not include those associated with anaesthesia for operations for ectopic pregnancy, ruptured uterus or abortion. The correct, total numbers for 1964–66 and 1967–69 have been calculated and included as additions in Table 19.2.

Table 19.6 *Direct, 'Associated', Indirect and Fortuitous deaths and deaths from cardiac disease (England and Wales, 1952–84*)*

Triennium	Direct deaths	'Associated' deaths	Indirect	Fortuitous	Percentage 'Associated'	Deaths from cardiac disease			Percentage 'Associated' due to Cardiac Disease
						Acquired	Congenital	Total	
1952–54	1094	316			22·4	121	NA	121	38·3
1955–57	861	339			28·3	102	NA	102	30·1
1958–60	742	254			25·5	66	NA	66	26·0
1961–63	692	244			26·1	68	13	71	29·1
1964–66	579	176			23·3	43	7	50	28·4
1967–69	445	218			32·9	34	15	49	22·5
LATE DEATHS EXCLUDED									
1970–72*	343	206			37·5	33	9	42	20·4
1973–75	227	111			32·8	14	4	18	16·2
1976–78	217	134	78	56	38·2	14	3	17	12·7
1979–81**	178**	121	90	31	40·5	12	4	16	13·2
1982–84	138	105	71	34	43·2	11	6	17	16·2

Source: Triennial CEMD Reports (England and Wales, 1952–54 to 1982–84)
* For changes in numbers and rates since last report see page 153.
**Includes two other Direct deaths omitted in last report.

During the second three triennia, between 1961–63 and 1967–69, the chapter headings began to assume their present form, but almost every report has seen change and innovation. Over these three triennia, the MMR per million from hypertensive diseases fell from 41.3 to 21.6 and haemorrhage fell from 36.5 to 16.7. The pulmonary embolism MMR fell from 51.2 to 30.5, but abortion remained almost unchanged, the comparable MMR being 55.1 and 47.6.

Deaths from anaesthesia, ectopic pregnancy and amniotic fluid embolism were relatively unchanged throughout these triennia, but sepsis and ruptured uterus fell sharply in 1967–69. Between 1961–63 and 1967–69 increasing provision of beds for hospital confinement, expansion of the maternity services and increasing expertise almost certainly contributed to the reduction in deaths from hypertensive diseases, pulmonary embolism and haemorrhage. The introduction in 1962 of a combined preparation of oxytocin and ergometrine for intramuscular use is a specific example of an advance which likely contributed to the reduction in postpartum haemorrhage deaths.

1970–72 to 1982–84

Table 19.5 shows the MMR by cause between 1970–72 and 1982–84 and demonstrates the effect of the 1967 Abortion Act which came into force in 1968. Abortion remained the main cause of death in 1970–72 despite a marked reduction in deaths from criminal and spontaneous abortion, because of an increase in deaths from therapeutic abortion. In retrospect, this proved to be associated with late abortions and with the use of major surgical techniques such as hysterotomy, often combined with sterilisation, or hysterectomy. By 1973–75, legal abortion was more often being performed earlier in pregnancy by simple vaginal methods and the abortion MMR began to fall rapidly. By 1982–84, MMR from abortion was very low and for the first time, there were no deaths from criminal abortion. The fall in the birth rate which began after 1964–66 (Table 19.1) was probably due mainly to the introduction of oral contraception and new intra-uterine contraceptive devices. However, sterilisation performed by laparoscopy, and legal abortion, must have also played an increasing part. These factors doubtless influenced the MMR by reducing grande multiparity and childbirth at extremes of reproductive age, as well as avoiding pregnancy in women with special medical risks. Changes in age and parity over the CEMD are shown in Tables 1.10 and 1.11.

Table 19.5 also shows that, compared with 1952–69, MMR from hypertensive diseases of pregnancy and pulmonary embolism fell relatively slowly between 1970 and 1984. They became equal main causes of death in 1982–84. Although the MMR from hypertensive diseases of pregnancy was lower in the 1980's than in the 1950's, the slow improvement over the last five triennia is disappointing. Recent CEMD have highlighted the serious hazards of hypertensive diseases of pregnancy, which can develop insidiously and progress rapidly to a dangerous stage with little warning. Death is still due mainly to cerebral haemorrhage, probably from failure to control severe hypertension, but can also result from disseminated intravascular coagulation, renal failure or liver necrosis. These complications can all be anticipated by appropriate investigations described in Chapter 2, in which it has again been suggested that expert teams should be established to advise about management in each region or take over cases if requested.

The MMR from pulmonary embolism remains a major problem, largely because no effective method has been developed for its prevention or early detection. The CEMD reports have repeatedly stressed the importance of an awareness of predisposing factors. The 1982–84 report has drawn attention to the hazards of pulmonary embolism in women delivered by elective Caesarean section for severe hypertensive diseases of pregnancy, as well as those with previous thromboembolism, those who are overweight or are treated by prolonged recumbency.

While the period between 1952 and 1969 was dominated by four main causes of death, the period between 1970 and 1984 has seen the emergence of other major causes of death. While hypertensive diseases of pregnancy and pulmonary embolism have remained of primary importance, complications of anaesthesia were the third main cause of death in 1979–81, followed by amniotic fluid embolism and ectopic pregnancy. In 1982–84, complications of anaesthesia were again the third commonest cause of death, followed by amniotic fluid embolism, abortion, haemorrhage and ectopic pregnancy.

Table 19.5 shows that the rate of fall in deaths from anaesthesia has been slow. A recently published report by the Association of Anaesthetists[4] expresses concern about this apparent lack of improvement. However, the MMR for anaesthesia calculated per million pregnancies is not a satisfactory index of the risk of death from this cause, because the number of anaesthetics administered in obstetric practice has increased considerably in recent years. Unfortunately, there are no reliable estimates of the number of anaesthetics adminstered to obstetric patients, but, an estimate can be obtained from the number of Caesarean sections which is known to have doubled over the last five triennia.

It is clear, however, that maternity patients have not been accorded the same standards of anaesthetic care as are generally available to surgical patients and much needs to be done to achieve this level of care in obstetric units. Although the rate of improvement in MMR from anaesthesia is probably greater than indicated by the rates in Table 19.5, each of the last five triennial reports has reported a high incidence of avoidable factors or substandard care in anaesthetic deaths.

While the MMR from genital sepsis and ruptured uterus has fallen markedly between 1970 and 1984, it is surprising that the MMR from ectopic pregnancy fell so much in 1982–84 when it had been unchanged over several previous triennia. The fall may have been due to improved diagnostic techniques including rapid β-HCG assay and improved ultrasound, or it may have been due to chance.

Amniotic fluid embolism shows no change in MMR. This is only to be expected since no advance has been made in its prevention, detection or treatment. The variability over the triennia in the MMR from Other Direct deaths, also classified as 'Miscellaneous', can be attributed to the difficulty of consistently attributing to this category, Direct deaths not attributable to other causes.

While Direct deaths from haemorrhage have fallen to their lowest level in 1982–84, haemorrhage contributes to many deaths attributed to other causes and remains a major factor in maternal mortality. This report, and that for 1978–82, have drawn particular attention to the anticipatable risk of severe haemorrhage at Caesarean section in women with one or more previous Caesarean sections in whom the placenta is anterior and low-lying. In such cases there is a high risk that the placenta will be pathologically adherent to the lower segment scar and that Caesarean section may have to be followed by hysterectomy. Another important development in the management of haemorrhage is the increasing reluctance of anaesthetists to provide an anaesthetic for manual removal of the placenta in the unfamiliar circumstances of, and the unfamiliar equipment associated with, a 'flying squad' call. Throughout England and Wales, 'flying squads' are tending to become mobile resuscitation units which initiate blood transfusion, ensure that the patient's condition is stable and then transfer her, under surveillance, to a fully equipped specialist unit where any necessary procedures can be performed with all facilities immediately available. These modified 'flying squads' are likely to remain of vital importance for the management not only of post partum haemorrhage of various types, but also for disorders such as spontaneous abortion with severe bleeding, antepartum haemorrhage, and severe, acute symptomatic hypertensive diseases of pregnancy.

Avoidable factors and substandard care

The incidence of avoidable factors in 1952–54 was approximately 45 per cent and by 1976–78, had increased to approximately 58 per cent. This increase more likely resulted from an increasingly high standard of assessment than from a deterioration in management. Since 1979–81, the term substandard care has been used instead of avoidable factors, with the object of taking into account not only failures in clinical care but also other factors which might have adversely affected care, including shortage of resources for the provision of staff and administrative failures in the service or in the provision of backup services such as anaesthesia, radiology or pathology. It has proved more difficult quantitatively to assess the incidence of substandard care but since many deaths are still associated with substandard care, further improvement must be possible.

Indirect, Fortuitous and 'Associated' Deaths

Table 19.6 lists the number of Direct and Indirect, Fortuitous, 'Associated' deaths and deaths from cardiac disease between 1952–54 and 1982–84. Up to 1973–75, deaths not directly due to pregnancy were classified as 'Associated' but since 1976–78 have been classified as Indirect or Fortuitous (Chapter 1). Since 1979–81, Late deaths, occurring between 42 days and one year after the end of pregnancy, have been included in a separate chapter in the reports. Deaths from cardiac disease have always been described in a separate chapter but up to 1973–75 were counted as 'Associated'. Since 1976–78 they have been counted as Direct, Indirect or Fortuitous deaths.

Table 19.6 shows that over the 11 triennia, Direct deaths have fallen more rapidly than Indirect or Fortuitous deaths so that the latter, the previous Associated deaths, have increased from around 25 per cent of all maternal deaths in the earlier triennia to around 40 per cent in the last three triennia. Clearly, greater attention will have to be paid to Indirect deaths in the future if the MMR is to continue to fall as rapidly as it has done in the past.

While the main causes of 'Associated' deaths in the earlier triennia were severe generalised infections or cardiac disease, recent reports have drawn attention to the increasing importance of disorders such as aneurysms (with death due to rupture of intracranial berry aneurysms, or to intra-abdominal rupture of aortic, splenic or other aneurysms, sometimes associated with Marfan's syndrome); to the tendency for epilepsy to worsen in pregnancy, causing death from uncontrolled convulsions and meriting better prophylaxis by administration of anticonvulsant agents in high enough doses to achieve therapeutic blood levels; of diabetes deteriorating, leading to death from various metabolic complications; and of respiratory failure in pregnancy associated with severe kyphosocoliosis.

Cardiac disease deaths have changed markedly in character over the 11 triennia and Table 19.6 shows how their contribution to 'Associated' deaths has lessened. The earlier years were dominated by many deaths from acquired heart disease, almost always of rheumatic origin. Because of many factors, including the effective use of penicillin for the treatment of rheumatic fever, the incidence of these disorders has fallen progressively and in the last four of five triennia, the MMR for acquired heart disease has more often been due to coronary atherosclerosis and myocardial infarction, or to cardiomyopathy, than to rheumatic carditis. Congenital heart disease is becoming increasingly important and recent CEMD reports have drawn attention to the hazards of pregnancy in women whose congenital heart disease has only partially been cured by surgery in childhood. Pre-pregnancy counselling is of vital importance for such women.

The maternal autopsy

Considering the importance of autopsy in establishing the often uncertain cause of maternal death, it is surprising it was not until 1979 that the introduction to the 8th report drew attention to the often unsatisfactory nature of autopsies in maternal deaths. The Advisor in Histopathology was invited to join the Central Assessors in Obstetrics and Anaesthetics in 1980 and Regional Assessors in Pathology were appointed in February 1981. Chapter 17 of this 1982–84 CEMD reports on progress. The quality of autopsy in maternal death has clearly been much improved and the Central and Regional Assessors in Pathology are to be congratulated on their success. Much remains to be done, however, and it is hoped that improvement will continue in the future.

Conclusions

Over the 11 triennia from 1952–54 to 1982–84, the CEMD for England and Wales have provided a unique audit of the effectiveness of the maternity services and served as a model for many other surveys. Especially in recent years, the enquiries have utilised more sophisticated statistical methodology. They have always been handicapped by a relative lack of comprehensive morbidity data, however, which makes it difficult to know if reduction in deaths from an individual cause has resulted from a reduced frequency of the disorder or from better management of a similar number of cases. Although maternal mortality is a much smaller problem now than it was 50 years ago, obstetricians will be interested to see that the *rate* of improvement in MMR has been constant since its first decline around 1937, halving every decade. During the 33 years of the CEMD MMR has fallen from 989 in 1951 to 86 in 1984, per million maternities. If MMR continues to decline at the same rate, maternal mortality for England and Wales in 1991 should be approximately 44 per million and in 2001 approximately 22 per million. When the new UK-based enquiries are published, they should permit comparisons with previous MMR for England and Wales, Scotland and Northern Ireland.

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Appendix A: Method of the Enquiry and important definitions

Method of the Enquiry 1982–84

In the case of a known or suspected maternal death, an enquiry was initiated by the District Medical Office (DMO), or Chief Administrative Medical Officer (CAMO) in Wales, of the district in which the woman was usually resident. An enquiry form (MCW97) was sent to general practitioners, midwives, health visitors, community physicians, consultant obstetricians and any other relevant staff who had been concerned with the care of the woman.

When all the information about the death had been collected, the DMO forwarded the form to the appropriate regional obstetric assessor. The regional anaesthetic assessor reviewed all cases who had received an anaesthetic. Every possible attempt was later made to obtain full details of any post mortem examinations and these were reviewed by the regional pathology assessors. The assesors added their comments and opinions regarding the cause or causes of death.

The MCW97 form was then sent to the Chief Medical Officer of the Department of Health and Social Security (DHSS) or the Chief Medical Officer of Wales. The Department's central assessors in obstetrics and gynaecology, anaesthetics and histopathology then reviewed all available recorded facts about each case and acted as final arbiters in assessing the many factors that may have led to death.

Strict confidentiality is observed at all stages of the Enquiry and the identity of the patient is erased from all forms, so that the opinions of assessors cannot be related to a named individual. After preparation of the Report all MCW97 forms are destroyed.

Definitions

Cause of death

A single *main* cause of death has been allotted and subsequently classified according to the International Classification of Diseases, Injuries and Causes of Death—ninth revision (ICD9) (see Tables A1, A2 and A3). This was not necessarily the immediate cause of death. Although deaths are assigned to one main cause, they may be referred to in other chapters, thus a death assigned to hypertensive disease of pregnancy, in which haemorrhage and anaesthesia also played a part, will be discussed in all three chapters.

Classification of maternal death

There is international agreement to subdivide causes of obstetric deaths into *Direct* and *Indirect*. ICD9 defines *Direct* obstetric deaths as 'those resulting from obstetric complications of the pregnant state (pregnancy, labour and puerperium), from interventions, omissions, incorrect treatment, or from a chain of events resulting from any of the above'. *Indirect* obstetric deaths are defined as 'those resulting from previous existing disease, or disease that developed during pregnancy and which was not due to direct obstetric causes, but which was aggravated by physiologic effects of pregnancy'. Only those deaths from other causes which happen to occur in pregnancy or the puerperium are excluded from maternal mortality as internationally defined. For the purposes of this Enquiry, these are defined as *fortuitous* deaths.

The numbers and rates of cases by cause of death for 1970–72 and 1973–75 are different from those quoted in the 1979–81 Report. They have been revised in line with the classification used in the present Report.

Conceptions and pregnancies

It is now customary for OPCS to publish annual estimates of conceptions. This is a count of maternities and legal terminations to mothers resident in England and Wales, adjusted to take account of the gestation recorded on the abortion notification.

An alternative denominator has been used for the computation of rates where the numerator includes deaths occurring early in pregnancy. These include abortions, ectopic pregnancies and anaesthesia. The number of pregnancies is estimated by adding the number of conceptions (see above) to the estimated number of ectopic pregnancies and spontaneous abortions admitted to NHS hospitals.

Expected deaths

The tables of successive reports of death rates per million maternities has facilitated comparisons with reports from earlier triennia. This has been continued in this report where the number of deaths is sufficient to give meaningful rates. Continued use has been made of the alternative technique first adopted in the 1979–81 report, whereby the number of *expected deaths* is calculated on the basis of age (or parity)—specific death rates for a total number of (previous) years (usually the period 1970–1981). This allows direct comparison with the *actual number of deaths* in the current triennium. It also facilitates the computation of the overall effects of factors such as age or parity on death rates for various causes of death by using an extended series of data over an extended period.

'Late' deaths

ICD9 defines a maternal death as 'the death of a woman while pregnant or within 42 days of termination of pregnancy, irrespective of the duration and the site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management but not from accidental or incidental causes'. This is in accord with the definition adopted by the International Federation of Gynaecology and Obstetrics (FIGO).

In the Confidential Enquiry, however, deaths have also been included if they occurred between 43 days and one year after delivery or abortion. These *'late' deaths* are now considered separately in Chapter 16.

Maternities

In this report deaths are usually related to the number of *maternities*. This is a count of the number of *mothers delivered* as distinct from the number of *babies born*; which, of course, include twins and other multiple births.

Recent changes in the calculations which provided numbers of maternities and pregnancies have led to changes in the data used as denominators in several tables.

Parity

Parity is defined as the number of previous pregnancies of 28 weeks gestation or more (regardless of the outcome of the pregnancy) *plus the present pregnancy*, whatever its duration. Birth Registration information about previous births is confined to legitimate births so that the construction of a parity distribution of all maternities (Appendix Table A6) requires the allocation of illegitimate births by parity plus some reallocation of legitimate births. This has been done by using statistics from the General Household Survey, a sample enquiry conducted each year by Social Survey Division of OPCS currently covering people in approximately ten thousand private households each year (one in two thousand of the population). Since 1979 the questionnaire used in this survey has included a question addressed to all single female respondents aged 18 to 49 years and all ever-married respondents aged 16 to 49 years asking them to state the dates of birth of all their children. In conjunction with answers to questions relating to the dates of any marriage and divorces, it is possible to construct a complete reproductive history or women in these age groups both within and outside marriage.

Substandard care

The term substandard care has been used to take into account failures in clinical care and also some of the underlying factors which may have produced a low standard of care for the patient. These include shortage of resources for staffing facilities and administrative failure in the maternity services and back-up facilities such as anaesthetic, radiological and pathology services. The term avoidable factors used previously was often misinterpreted to mean that avoiding these factors would have prevented the death.

Table A1 *Direct maternal deaths. Deaths due to or associated with pregnancy and childbirth, England and Wales, 1982–84*

ICD No	Cause of death	Assigned Code	OPCS Code	'Late' Deaths	
				Assigned Code	OPCS Code
II	**Neoplasms**				
181	Malignant neoplasm of placenta	1	1	–	–
IV	**Diseases of blood and blood-forming organs**				
283	Acquired haemolytic anaemias	–	1	–	–
VI	**Diseases of the nervous system and sense organs**				
348	Other conditions of brain	–	2	–	–
VII	**Diseases of the circulatory system**				
415	Acute pulmonary heart disease	–	4	–	–
425	Cardiomyopathy	–	–	–	1
428	Heart failure	–	1	–	–
430	Subarachnoid haemorrhage	–	1	–	–
431	Intracerebral haemorrhage	–	3	–	–
433	Occlusion and stenosis of precerebral arteries	–	1	–	–
434	Occlusion of cerebral arteries	–	1	–	–
X	**Diseases of the genitourinary system**				
580	Acute glomerulonephritis	–	1	–	–
611	Other disorders of breast	–	1	–	–
XI	**Complications of pregnancy, childbirth and the puerperium**				
633	Ectopic pregnancy	10	10	–	–
634	Spontaneous abortion	4	2	1	1
635	Legally induced abortion	7	5	–	–
637	Unspecified abortion	–	1	–	–
639	Complications following abortion and ectopic and molar pregnancies	–	1	–	–
641	Antepartum haemorrhage, abruptio placentae, and placenta praevia	4	7	–	–
642	Hypertension complicating pregnancy, childbirth and the puerperium	25	16	1	1
643	Excessive vomiting in pregnancy	1	1	–	–
646	Other complications of pregnancy, not elsewhere classified	9	6	–	–
648	Other current conditions in the mother classifiable elsewhere but complicating pregnancy, childbirth and the puerperium	–	1	–	–
653	Disproportion	–	1	–	–
656	Other fetal and placental problems affecting management of mother	–	1	–	–
658	Other problems associated with amniotic cavity and membranes	1	1	–	–
660	Obstructed labour	–	1	–	–
661	Abnormality of forces of labour	–	1	–	–
665	Other obstetrical trauma	5	7	–	–
666	Postpartum haemorrhage	3	4	–	–
668	Complications of the administration of anaesthetic or other sedation in labour and delivery	18	6	1	1
669	Other complications of labour and delivery, not elsewhere classified	–	4	–	–
670	Major puerperal infection	1	1	–	–

Table A1—*continued*

ICD No	Cause of death	Assigned Code	OPCS Code	'Late' Deaths Assigned Code	'Late' Deaths OPCS Code
671	Venous complications in pregnancy and the puerperium	3	8	–	1
673	Obstetrical pulmonary embolism	40	24	2	–
674	Other and unspecified complications of the puerperium, not elsewhere classified	5	2	1	1
675	Infections of the breast and nipple associated with childbirth	1	–	–	–
XIV	**Congenital anomalies**				
747	Other congenital anomalies of circulatory system	–	1	–	–
XVI	**Symptoms, signs and ill-defined conditions**				
799	Other ill-defined and unknown causes of morbidity and mortality	–	1	–	–
XVII	**Injury and poisoning**				
852	Subarachnoid, subdural and extradural haemorrhage, following injury	–	1	–	–
	External causes of injury and poisoning				
E 870*	Accidental cut, puncture, perforation or haemorrhage during medical care	–	1	–	–
E 876*	Other and unspecified misadventures during medical care	–	6	–	–
Total		138	138	6	6

*Although the E codes were intended for use as an additional classification of causes assigned to rubrics in Chapters I to XVII (predominantly Chapter XVII), this practise has not been observed here. Thus, an E code appearing in the column headed "OPCS Code" will not be double-coded anywhere else in the column, although it will be coded elsewhere in the column headed "Assigned Code".

Table A2 *Indirect maternal deaths. England and Wales, 1982–84*

ICD No	Cause of death	Assigned Code	OPCS Code	'Late' Deaths Assigned Code	'Late' Deaths OPCS Code
I	**Infectious and parasitic diseases**				
034	Streptococcal sore throat and scarlatina	–	1	–	–
II	**Neoplasms**				
174	Malignant neoplasm of female breast	–	1	–	1
180	Malignant neoplasm of cervix uteri	–	–	–	2
VI	**Diseases of the nervous system and sense organs**				
325	Phlebitis and thrombophlebitis of intracranial venous sinuses	–	1	–	–
345	Epilepsy	–	3	–	–

Table A2—*continued*

ICD No	Cause of death	Assigned Code	OPCS Code	'Late' Deaths	
				Assigned Code	OPCS Code
VII	**Diseases of the circulatory system**				
396	Diseases of mitral and aortic valves	–	1	–	–
410	Acute myocardial infarction	–	6	–	–
425	Cardiomyopathy	–	–	–	1
429	Ill-defined descriptions and complications of heart disease	–	–	–	1
430	Subarachnoid haemorrhage	–	4	–	1
431	Intracerebral haemorrhage	–	2	–	–
433	Occlusion and stenosis of precerebral arteries	–	1	–	–
VIII	**Diseases of the respiratory system**				
481	Pneumococcal pneumonia	–	1	–	–
507	Pneumonitis due to solids and liquids	–	–	–	1
X	**Diseases of the genitourinary system**				
593	Other disorders of kidney and ureter	–	1	–	–
XI	**Complications of pregnancy, childbirth and the puerperium**				
635	Legally induced abortion	–	1	–	–
642	Hypertension complicating pregnancy, childbirth and the puerperium	–	2	1	1
646	Other complications of pregnancy, not elsewhere classified	–	1	–	–
647	Infective and parasitic conditions in the mother classifiable elsewhere but complicating pregnancy, childbirth and the puerperium				
.6	Other viral diseases	1	–	–	–
.8	Other specified infective and parasitic diseases	3	–	–	–
648	Other current conditions in the mother classifiable elsewhere but complicating pregnancy, childbirth and the puerperium				
.0	Diabetes mellitus	3	1	–	–
.2	Anaemia	2	1	–	–
.4	Mental disorders	7	1	6	–
.5	Congenital cardiovascular disorders	5	2	–	–
.6	Other cardiovascular diseases	11	5	2	–
.9	Other	24	13	6	2
656	Other fetal and placental problems affecting management of mother	–	1	–	–
666	Postpartum haemorrhage	–	1	–	–
671	Venous complications in pregnancy and the puerperium	–	2	–	–
674	Other and unspecified complications of the puerperium, not elsewhere classified	15	7	2	1
XIV	**Congenital anomalies**				
745	Bulbus cordis anomalies and anomalies of cardiac septal closure	–	2	–	–
746	Other congenital anomalies of heart	–	1	–	–
	External causes of injury and poisoning				
E 858	Accidental poisoning by other drugs	–	1	–	–
E 875	Contaminated or infected blood, other fluid, drug or biological substance	–	1	–	–

Table A2—*continued*

ICD No	Cause of death	Assigned Code	OPCS Code	'Late' Deaths	
				Assigned Code	OPCS Code
E 878	Surgical operation and other surgical procedures as the cause of abnormal reaction of patient, or of later complication, without mention of misadventure at the time of operation	–	1	–	–
E 950	Suicide and self-inflicted poisoning by solid or liquid substances	–	1	–	1
E 952	Suicide and self-inflicted poisoning by other gases and vapours	–	1	–	–
E 953	Suicide and self-inflicted injury by hanging, strangulation and suffocation	–	–	–	2
E 954	Suicide and self-inflicted injury by submersion (drowning)	–	1	–	–
E 958	Suicide and self-inflicted injury by other and unspecified means	–	1	–	3
E 987	Falling from high place, undetermined whether accidentally or purposely inflicted	–	1	–	–
Total		71	71	17	17

Table A3 *Fortuitous deaths England and Wales, 1982–84*

ICD No	Cause of death	Assigned Code	OPCS Code	'Late' Deaths	
				Assigned Code	OPCS Code
I	**Infectious and parasitic diseases**				
018	Miliary tuberculosis	1	2	–	–
038	Septicaemia	–	–	1	1
052	Chickenpox	1	1	–	–
054	Herpes simplex	–	–	1	–
074	Specific diseases due to Coxsackie virus	–	–	–	1
136	Other and unspecified infectious and parasitic diseases	–	–	–	1
II	**Neoplasms**				
151	Malignant neoplasm of stomach	1	1	–	–
153	Malignant neoplasm of colon	–	–	1	1
154	Malignant neoplasm of rectum, rectosigmoid junction and anus	–	–	1	1
155	Malignant neoplasm of liver and intrahepatic bile ducts	1	1	–	–
157	Malignant neoplasm of pancreas	1	1	1	1
162	Malignant neoplasm of trachea, bronchus and lung	2	2	–	–
172	Malignant melanoma of skin	–	–	1	1
189	Malignant neoplasm of kidney and other and unspecified urinary organs	2	2	–	–
191	Malignant neoplasm of brain	5	3	2	–
199	Malignant neoplasm without specification of site	–	–	3	3
200	Lymphosarcoma and reticulosarcoma	–	–	2	1

Table A3—*continued*

ICD No	Cause of death	Assigned Code	OPCS Code	'Late' Deaths	
				Assigned Code	OPCS Code
202	Other malignant neoplasm of lymphoid and histiocytic tissue	–	–	1	1
208	Leukaemia of unspecified cell type	–	–	–	1
212	Benign neoplasm of respiratory and intrathoracic organs	1	–	–	–
239	Neoplasm of unspecified nature	–	–	–	2
III	**Endocrine, nutritional and metabolic diseases and immunity disorders**				
241	Nontoxic nodular goitre	1	1	–	–
276	Disorders of fluid, electrolyte and acid-base balance	–	–	1	1
IV	**Diseases of blood and blood-forming organs**				
284	Aplastic anaemia	1	–	–	–
287	Purpura and other haemorrhagic conditions	1	–	–	–
288	Diseases of white blood cells	–	–	1	–
VI	**Diseases of the nervous system and sense organs**				
320	Bacterial meningitis	2	1	–	–
323	Encephalitis, myelitis and encephalomyelitis	–	–	1	–
331	Other cerebral degeneration	–	–	–	1
340	Multiple sclerosis	–	1	–	–
345	Epilepsy	1	1	1	1
VII	**Diseases of the circulatory system**				
410	Acute myocardial infarction	2	1	2	2
422	Acute myocarditis	–	–	2	–
429	Ill-defined descriptions and complications of heart disease	–	–	–	1
430	Subarachnoid haemorrhage	–	–	4	4
431	Intracerebral haemorrhage	–	–	1	2
446	Polyarteritis nodosa and allied conditions	–	–	1	–
482	Other bacterial pneumonia	–	–	–	1
485	Bronchopneumonia, organism unspecified	1	–	–	–
487	Influenza	–	–	–	1
493	Asthma	–	–	3	3
515	Post-inflammatory pulmonary fibrosis	–	–	–	1
IX	**Diseases of the digestive system**				
552	Other hernia of abdominal cavity with obstruction, without mention of gangrene	–	–	1	–
X	**Diseases of the genitourinary system**				
580	Acute glomerulonephritis	–	–	–	1
592	Calculus of kidney and ureter	–	–	1	1
614	Inflammatory disease of ovary, fallopian tube, pelvic cellular tissue and peritoneum	–	–	1	1
XI	**Complications of pregnancy, childbirth and the puerperium**				
637	Unspecified abortion	–	1	–	–

Table A3—*continued*

ICD No	Cause of death	Assigned Code	OPCS Code	'Late' Deaths	
				Assigned Code	OPCS Code
648	Other current conditions in the mother classifiable elsewhere but complicating pregnancy, childbirth and the puerperium				
.2	Anaemia	—	1	—	—
.6	Other cardiovascular diseases	—	1	—	—
.9	Other	—	3	—	—
XIII	**Diseases of the musculoskeletal system and connective tissue**				
710	Diffuse diseases of connective tissue	—	—	2	1
XIV	**Congenital anomalies**				
742	Other congenital anomalies of nervous system	—	—	1	—
XVII	**Injury and poisoning**				
869	Internal injury to unspecified or ill-defined organs	1	—	—	—
994	Effects of other external causes	—	—	2	—
	External causes of injury and poisoning				
E 812	Other motor vehicle traffic accident involving collision with another motor vehicle	—	—	1	1
E 814	Motor vehicle traffic accident involving collision with pedestrian	2	2	—	—
E 815	Other motor vehicle traffic accident involving collision on the highway	—	1	—	—
E 841	Accident to powered aircraft, other and unspecified	—	—	1	1
E 867	Accidental poisoning by gas distributed by pipeline	—	—	2	2
E 882	Fall from or out of building or other structure	—	—	1	1
E 910	Accidental drowning and submersion	—	—	—	1
E 911	Inhalation and ingestion of food causing obstruction of respiratory tract or suffocation	1	1	—	—
E 950	Suicide and self-inflicted poisoning by solid or liquid substances	1	1	3	3
E 953	Suicide and self-inflicted injury by hanging, strangulation and suffocation	—	—	1	1
E 957	Suicide and self-inflicted injuries by jumping from high place	—	—	1	1
E 965	Assault by firearms and explosives	—	—	1	1
E 968	Assault by other and unspecified means	4	2	—	—
E 983	Hanging, strangulation or suffocation, undetermined whether accidentally or purposely inflicted	—	—	—	1
E 988	Injury by other and unspecified means, undetermined whether accidentally or purposely inflicted	—	2	—	—
Total		34	34	50	50

The footnote to Table A1 regarding E codes applies here but in some cases a death has been assigned the same E code in both columns

Table A4 *Number of deaths by age of mother in the Enquiry 1982–84 compared with those included in 1979–81 and the rate per million maternities*

Age (years)	Number of maternal deaths						Rates per million maternities					
	1979–81			1982–84			1979–81			1982–84		
	Direct	Indirect	Fortuitous	Direct	Indirect	Fortuitous	Direct	Indirect	Fortuitous	Direct	Indirect	Fortuitous
Under 16	–	–	–	–	1	–	90.4	28.3	17.0	54.8	42.6	24.4
16–17	5	2	1	2	2	–						
18–19	11	3	2	7	4	4						
20–24	34	29	4	27	16	7	57.8	49.3	6.8	47.0	27.9	12.2
25–29	49	20	11	39	19	12	74.4	30.4	16.7	60.9	29.7	18.7
30–34	41	27	8	28	13	5	107.9	71.0	21.0	77.4	35.9	13.8
35–39	27	5	4	27	13	6	272.5	50.5	40.4	220.7	106.3	49.1
40–44	11	4	–	8	3	–	599.9	218.1	–	482.4	160.6	–
45+	–	–	1	–	–	–	–	–	535.6	–	–	–
Total	178	90	31	138	71	34	92.5	46.8	16.1	73.3	37.7	18.0

Table A5 *Number of deaths by parity of mother in the Enquiry 1982–84 compared with those included in 1979–81 and the rate per million maternities*

Parity	Number of maternal deaths						Rates per million maternities					
	1979–81			1982–84			1979–81			1982–84		
	Direct	Indirect	Fortuitous	Direct	Indirect	Fortuitous	Direct	Indirect	Fortuitous	Direct	Indirect	Fortuitous
1	69	36	10	56	26	11	87.9	45.8	12.7	73.4	34.1	14.4
2	54	31	12	32	23	11	79.8	45.8	17.7	49.0	35.2	16.8
3	26	11	3	26	11	3	88.2	37.3	10.2	88.8	37.6	10.3
4	16	9	–	16	7	5	152.9	86.0	–	149.2	65.3	46.6
5–9	9	2	4	8	4	3	143.1	31.8	63.6	118.7	59.3	44.5
10 or more	–	–	–	–	–	–	–	–	–	–	–	–
Not stated	4	1	2	–	–	1	–	–	–	–	–	–
Total	178	90	31	138	71	34	92.5	46.8	16.1	73.3	37.7	18.0

Table A6 *True birth order—maternities 1970–84, England and Wales*

Years	True Birth Order*	Age (years)							
		Total	Under 20	20–24	25–29	30–34	35–39	40–44	45+
1979–81	1	785,203	145,199	303,556	235,286	85,148	14,017	1,860	137
	2	676,350	28,700	209,934	264,863	142,932	26,159	3,563	199
	3	294,626	2,717	58,127	106,748	96,708	26,727	3,402	197
	4	104,658	344	13,907	35,763	34,139	17,123	3,182	200
	5+	62,888	16	3,213	16,005	21,144	15,046	6,330	1,134
Total		1,923,725	176,976	588,737	658,665	380,071	99,072	18,337	1,867
1982–84	1	762,961	137,099	292,700	228,665	84,323	18,101	1,925	147
	2	653,502	24,365	203,508	253,989	131,727	35,772	3,949	192
	3	292,666	2,395	58,777	101,307	92,000	33,992	3,977	219
	4	107,223	377	15,181	38,777	30,916	18,595	3,155	222
	5+	67,402	21	4,014	17,786	22,957	15,858	5,670	1,096
Total		1,888,753	164,256	574,180	640,524	361,923	122,318	18,675	1,877
1970–84	1	3,906,058	778,940	1,564,745	1,143,632	337,636	69,513	10,860	741
	2	3,423,320	164,062	1,155,222	1,368,922	582,911	131,362	19,828	1,013
	3	1,482,136	16,651	329,364	561,762	416,127	133,788	23,107	1,338
	4	563,653	1,805	83,035	199,460	171,278	89,449	17,512	1,114
	5+	400,932	115	19,513	101,161	128,925	103,947	41,923	5,348
Total		9,776,098	961,572	3,151,879	3,374,937	1,636,877	528,059	113,229	9,545

1981 figures are based on a 10% sample for age-group and parity.

*Previous live births regardless of legitimacy. Birth registration figures adjusted using information from the 1979–82 General Household Survey. Adjustment factors derived from the 1979–82 GHS have been applied to all years shown in this table to convert live births to estimated maternities.

Table A7 *Number of Direct maternal deaths included in the Enquiry 1982–84 by age and parity*

Age (years)	Parity							
	1	2	3	4	5–9	10+	Not stated	All
Under 16	–	–	–	–	–	–	–	–
16–17	2	–	–	–	–	–	–	2
18–19	7	–	–	–	–	–	–	7
20–24	16	7	4	–	–	–	–	27
25–29	21	9	7	2	–	–	–	39
30–34	7	9	6	4	2	–	–	28
35–39	3	7	7	8	2	–	–	27
40–44	–	–	2	2	4	–	–	8
Not stated	–	–	–	–	–	–	–	–
Total	56	32	26	16	8	–	–	138

*For definition of parity see page 154

Table A8 *Death rate of Direct maternal deaths per million maternities by age and parity* 1982–84, England and Wales*

Age (years)	Parity					Rates/1,000,000
	1	2	3	4	5+	Total
Under 20	65.6	–	–	–	–	54.8
20–24	54.7	34.4	68.1	–	–	47.0
25–29	91.8	35.4	69.1	51.6	–	60.9
30–34	83.0	68.3	65.2	129.4	87.1	77.4
35–39	165.7	195.7	205.9	430.2	126.1	220.7
40+	–	–	476.6	592.2	591.2	389.3
Total	73.4	49.0	88.8	149.2	118.7	73.3

*For definition of parity see page 154

Appendix B Acknowledgements

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